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THE PRINCIPLES OF DIAGNOSIS.[1]

By **F. Guy Griffiths, M.D.,**
Honorary Physician, Royal North Shore Hospital, Sydney.

IT is wise from time to time to escape from routine where often we cannot see the wood for the trees and to contemplate principles in a general survey of our subject.

By diagnosis we understand the recognition of disease and I propose to invite you to consider, not methods of examination, but the principles by which we arrive at a diagnosis from the results observed in an examination.

There is no need to dilate on the value of diagnosis; all admit *qui bene diagnoscit, bene curat.* Yet, strangely enough, text-books devote much space to methods of examination and little or none to the process of logical reasoning by which the findings are interpreted.

In distinguishing diseases we may be content with various degrees of exactness. We may note merely some deformity or loss of power; we may go further and discover the particular structure at fault, thus making an anatomical diagnosis; or further still and recognize the character, as well as the situation, of the lesion, making a pathological diagnosis. Our power to accomplish this we owe in a large extent to Laennec, that glory of French medicine. Or, proceeding still further, we may, following the precept of Lucretius *causas cognoscere rerum,* elucidate the problem to its utmost depth and discover the nature of the agent causing the lesion, thus making an ætiological diagnosis.

For the successful accomplishment of this process there are several *desiderata*: (*i.*) careful observation, (*ii.*) a sound knowledge of anatomy and of physiology, normal and morbid, that is to say, of general pathology in its fullest and widest sense, and (*iii.*) above all, a capacity for critical judgement. Only a true sense of proportion in estimating the values of different data will enable one to weigh matters and arrive at a sound decision. Some authorities think that this faculty is rarely vouchsafed to any but masculine minds.

So much then for the varieties and the requirements of diagnosis. We come now to the methods. The most powerful is that of exhaustive analysis. After a careful investigation of the medical history and of the present state of the patient, entailing a thorough examination by clinical and laboratory means, one makes a comprehensive survey of all the possibilities, that is to say, infers all the possible causes of the collected data. It is important to note

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on September 30, 1921. This paper was submitted to the Australasian Medical Congress, Brisbane, 1920, and has been published in abstract in the *Transactions* of the Congress.

that the process by which causes are suggested, is inductive and not deductive; it is in fact the essential method of scientific investigation used by the ancient Greek philosophers, systematized by Aristotle and elaborated to its full perfection by our own Francis Bacon.

Inferences will usually be suggested by clinical knowledge, but may be by other considerations, *e.g.*, epidemiological and therapeutic. The therapeutic suggestions in diagnosis are sometimes of great interest: the most commonly offered examples are iodine in doubtful syphilis and quinine in fevers. I remember a patient in the Australian Imperial Force (Egypt) whose illness had been diagnosed as malaria, but who grew worse under quinine and manifested severe cinchonism; this was in such striking contrast with the almost invariable absence of ill-effects from quinine in malarial patients, though taking such enormous doses as two or three grammes daily, that I reviewed the diagnosis, stopped the quinine and disclosed typical enteric, one of the two cases among inoculated soldiers that I saw in two years. Similarly, the failure of anti-dysenteric serum and of emetine in dysentery sometimes led to the discovery of malarial enteritis.

Other inferences may be suggested by the possibilities of notorious simulation, *e.g.*, of phthisis by hydatid of the lung and of Bright's disease by tumour of the brain.

Sometimes it will be possible to confirm or exclude inferences by the presence or absence of signs deduced from them and it is here that laboratory reactions find their proper use, for they may afford conclusive evidence in the form of pathognomonic signs.

But whilst laboratory tests are often of the greatest importance, we must beware lest they lead us astray, as they surely will if we attach to them importance beyond their worth. This will happen if we allow those who ought to know better, to describe a non-specific reaction as specific, a mistake which is habitually made in the case of Wassermann's test; or, again, if we fail to realize that a sign of this kind may be present in a person who is infected, but not affected, one who harbours a parasite, but is not diseased. Without due care a physician is liable, if he depend too much on laboratory diagnosis, to discover alleged, but absent, disease. Again, too, it not infrequently happens that, while a patient affords laboratory evidence of one disease, his immediate and urgent symptoms are due to another; the victim of chronic, mild tuberculosis, syphilis or malaria continues liable to any of the numerous ills to which flesh is heir.

It cannot be too frequently and impressively stated that a firm reliance upon clinical principles is of the greatest value in guarding us from such dire errors.

In making inferences, caution is necessary to avoid various possible fallacies.

It is a truism to say that judgement should be suspended until the completion of the examination and that one should not diagnose from symptoms alone; yet there is at least one disease in which we may fail to discover confirmatory signs and be compelled to rely solely on the patient's history of her symptoms. I refer to gastric ulcer.

Another fallacy to be avoided is the diagnosis of two diseases where one only is present. We must obey the law of parsimony of hypotheses and be guided by the "razor" of William of Occam, *entia non sunt præter necessitatem multiplicanda*. Following our usual practice of disparaging men of our own race and lauding foreigners, many of us ignore this great mediæval Englishman.[1]

Of course, we must not go to extremes, but must recognize that it is not unknown for a patient to suffer from two or more different diseases. We may sometimes feel that it is only our own limitations which prevent us discovering one lesion capable of causing all the signs and symptoms present, but at other times the patient who describes himself as "suffering from a complication of diseases" is undoubtedly correct. This is particularly so in the case of chronic infections on which others may supervene. Thus, chronic pulmonary tuberculosis is frequently complicated by a bronchitis due to streptococcus, pneumococcus, *Bacillus influenzæ* or other bacterium. Indeed, any other infection may occur in its course. I have known typhoid, malaria and measles do so. The frequency with which pulmonary tuberculosis is contracted by those with long-standing pneumonokoniosis or diabetes, is familiar to all and I have seen it associated with hydatid of the lung. Perhaps the most extraordinary instance of this nature I can remember is that of a lady whose case I have recorded elsewhere, who had tubercle bacilli in her expectoration, a gastric ulcer with violent hæmatemesis, a tapeworm, a retroversion of the uterus and, while suffering from all these, fell ill of an acute fever during an epidemic of typhoid.

In Palestine double infections by malaria and influenza were common and sometimes diphtheria was added. I have seen both tuberculous and cancerous ulcers in the same colon.

We must be ever alert to recognize newly described diseases, to distinguish an occasional patient with myxœdema among a group suffering from chronic Bright's disease, to separate paratyphoid A and B from enteric and not to overlook *encephalitis lethargica*. Cautious self-restraint is often required to avoid the fallacy of diagnosing the disease which happens to be fashionable because some great one has recently suffered from it or because prominent practitioners are obsessed by its alleged prevalence.

It is necessary to exercise care to distinguish entirely different diseases requiring entirely different treatment, but yet so similar in their most striking manifestations as to simulate one another closely. A typical example is that of tumour of the brain and chronic nephritis. During an epidemic of bubonic plague one may see cases of diffuse diplococcal infection exhibiting a hæmorrhagic periadenitis liable to deceive the very elect. Where tropical dys-

[1] William of Ockham or Occam was the fifth of the six great British schoolmen—a galaxy such as no other nation can boast—John Scotus Eriugena, John of Salisbury, Roger Bacon, Duns Scotus, William of Occam and Wycliffe, the leader of the Reformation.

entery is common, cancer of the rectum may escape notice.

Refinements in diagnosis which separate out subclasses from classes, may become of extreme importance in treatment. Thus, during the recent campaign in Palestine, it was necessary to distinguish among dysenteries the amœbic, the bacillary, the malarial, the flagellate and so on. So, too, among ulcerations of the pharynx; diphtheria and Vincent's angina required treatment very different from that of follicular tonsillitis.

It is conceivable that it may become important in therapeutics to distinguish not only paratyphoid A and B from typhoid, but infections by the four groups of pneumococcus one from another.

Yet refinements in diagnosis may easily be carried to extremes. It is rarely of value to a patient to be able to estimate the exact extent of a tuberculous invasion of the lung and in cases of genito-urinary tuberculosis where one employs constitutional treatment, it imports little to the patient whether the main lesion be epididymal, renal, ureteral or vesical.

In estimating the claims of rival inferences as the possible diagnosis there are two main lines of approach: (*i.*) diagnosis by exclusion and (*ii.*) differential diagnosis. The first we owe to the French physicians of the early part of last century; the classical examples are afforded by the work of Laennec, of Louis and of Velpeau. It was exhibited in a high degree of excellence by Trousseau. The method consists in considering one by one all possible inferences and one by one excluding those which further investigation shows to be inadequate or unlikely, till only one, the most probable, remains for acceptance.

This, beyond doubt, is the method *par excellence*. But it is difficult, tedious and cumbersome.

If symptoms invariably present in a particular disease are absent or if symptoms incompatible with this disease—which, of course, can rarely happen—are present, the disease may be certainly excluded.

In the method of differential diagnosis the preliminary stages of the process are the same, but in place of considering the hypotheses one by one and excluding the unlikely, one compares them all and chooses the most likely.

The method is facilitated by the employment of tables, often very elaborately constructed, to the compilation of which some authors have devoted extraordinary labour and ingenuity.

Despite every precaution, there will remain a residuum of undiagnosable cases, partly because there are diseases which even the greatest masters have not yet distinguished, much less described; partly because different diseases are not sharply defined one from another, so as to be capable of separate pigeon-holing, but merge gradually one into another, being better represented as more or less closely aggregated in the neighbourhood of distinct types, but some discretely scattered in outlying positions, so that one cannot say to which type they belong; and, finally, because a patient sometimes dies before the characteristic symptoms of his disease have developed, so that even *post mortem* it may be impossible to identify it.

If there are some cases in which diagnosis is impossible, naturally there are many more in which it remains uncertain, so that the best one can do is to choose the more common and probable. Thomas Young, an English physician of one hundred years ago, famous as the developer of the undulatory theory of light, gave offence to many colleagues by insisting that all diagnosis was simply a matter of balancing probabilities, that it is seldom that the evidence is conclusive. So much from this being a weak feature, it might become a source of strength, for if the frequency of various symptoms in various diseases were accurately tabulated, it would become possible to calculate the probability of each of the various possible causes, by ordinary arithmetic according to the rules of the calculus of probabilities for the case of *a posteriori* probability. However, the prospect of applying this method remains far distant.

I have said that the most powerful method of diagnosis is that of exhaustive analysis and afterwards of either exclusion or differentiation. It is the method usually recommended in text-books, but it is not the method usually adopted in practice, for not only the tiro but also most experts employ the method of physiognomic diagnosis by direct recognition, that is to say, they recognize diseases in the same way as they recognize acquaintances, because they have seen them before and know their features, even though they are unable to explain how or why they know them. I do not mean to say that the physician could give no better reason for his faith that a particular case was one of a particular disease than could the man in the street for the recognition of his friend, for the physician does analyse features to a certain extent; perhaps his recognition should be compared rather with that of the detective, who not only knows a criminal by sight, but knows also his height and build, the shape of his nose and ears, the colour of his eyes and hair.

The faculty for correctly recognizing diseases is acquired only as the result of long and earnest study. It demands a wide knowledge of general pathology and a close acquaintance with the natural history of diseases, which will enable one to avoid unnecessary labour in the investigation of non-essentials and to concentrate attention on what is important in the case before one, but when exercised by a master of the art, such as the late Sir Samuel Wilks or the late Dr. William Cayley, it appears to the onlooker to be instinctive and instantaneous rather than discriminative. In the hands of the ordinary practitioner it is less brilliant and more laborious; he may at times make a successful "spot" or "snap" diagnosis, but he will be very foolish if he neglects to verify it by confirmatory and, if possible, by conclusive tests. Usually his nearest approach to the physiognomic method will be to decide quickly that the case falls within a group of diseases which present the same common features, and then to exclude one by one those of the group which are more important in prognosis or treatment. If he be then left with several alternatives between which it is unimportant to distinguish, he will generally be content. If he be demonstrating to students, or if he be ambitious to cultivate his own powers, he will, of course, proceed further.

OBSERVATIONS ON ANOXÆMIA, WITH AN ACCOUNT OF AN ACUTE MEDICAL EMERGENCY IN INFANTS DUE TO OXYGEN WANT.[1]

By W. F. Litchfield, M.B.,
Honorary Physician, Royal Alexandra Hospital for Children, Camperdown.

IN the *Australasian Medical Gazette*, October 30, 1910, Dr. E. S. Littlejohn reported a case of apnœa in an infant one month old. The child, after a slight attack of bronchitis, suffered from periodic breathing and attacks of apnœa. In all, artificial respiration was performed seventy-four times. The breathing was noticed to be shallow throughout. Oxygen inhalation and mustard baths were the chief therapeutic measures. The child recovered.

Since that time several cases of a similar character have come under my notice. In the majority of my cases the disturbance started with bronchitis of a mild kind. I have also seen young children with whooping cough show like symptoms. The pathology of the condition was a puzzle to me until I read J. S. Haldane's article on anoxæmia in the *British Medical Journal* of July 19, 1919, when it became reasonably clear that the condition was due to anoxæmia or oxygen want in the young subjects.

I shall briefly record two cases that have come under my notice recently and then discuss them in the light of Haldane's article:

W.R., a male infant, aged eight weeks, was admitted to the Children's Hospital on June 4, 1921. He had been ill for two days, suffering from a cough. On examination, he was found to be a well-nourished, breast-fed infant. The breathing was rapid and shallow and there was some cyanosis. The temperature was not raised and ranged from 35.5° C. to 36.5° C. throughout his illness. In the ward his breathing became periodic, of a Cheyne-Stokes's type, and at 4.30, 4.45, 6.0 and 8.0 p.m. his breathing ceased entirely. The periods of apnœa lasted from a few seconds to sixty seconds or longer. A few scattered rhonchi were detected in the lungs, but the bronchitis was not of a severe order. The breathing remained irregular all night, with varying degrees of cyanosis. Next day oxygen was applied continuously by means of a funnel and tube. The child's colour improved and the breathing became more regular. Next day the breathing became increasingly shallow and the child passed quietly away. The respirations averaged 52 and the pulse-rate 150 to the minute. Dr. W. J. Hull, my House Physician, reported a slowing of the pulse during the apnœic attacks.

The other patient was a baby admitted a few days earlier under the care of Dr. E. S. Littlejohn, who has allowed me to refer to it. This was a boy, aged seven weeks, who came to the Hospital collapsed and cyanosed. He revived after a mustard bath, but the temperature remained subnormal throughout. Complete cessation of breathing occurred at frequent intervals during the day and night, in spite of the continuous application of oxygen, and the child died quietly twenty hours after admission. The child was well nourished and was not suffering in any way other than from a slight form of bronchitis.

These cases constitute a very definite clinical group, comprising infants not older than two or three months, who, without apparent cause or following a slight bronchial catarrh, develope periodic breathing, culminating in alarming attacks of apnœa, a varying amount of cyanosis and a liability to a quiet death succeeding a term of rapid, shallow breathing.

The respiration or breathing in health and disease is under three main influences:

(*i.*) Reflex nervous action.
(*ii.*) Carbon dioxide tension in the arterial blood.
(*iii.*) Oxygen supply.

The respiratory centre in the medulla may be disturbed by influences from above, such as the will and emotion, and by reflex nervous actions from the periphery. Stimulation of almost any afferent nerve of the body may influence breathing, but the special afferent nerve of respiration is the vagus. A positive ventilation of the lung, implying an increase of intra-alveolar pressure of gas, inhibits inspiration and stimulates expiration, while a negative ventilation stimulates inspiration and inhibits expiration. This would seem to be the mechanism which keeps up the normal swing of breathing. The same mechanism concerns clinical phenomena. A narrowing of the air passages from any cause induces a negative ventilation of the lungs, because when the chest expands on inspiration, the free entry of air is impeded. Dyspnœa or difficulty of breathing is the result. The action of the respiratory centre is usually enhanced at the same time, owing to a retention of carbon dioxide in the blood. The exaggerated respiratory efforts in dyspnœa, which are mainly inspiratory, I take it, are called forth reflexly by a negative pressure in the lungs. I draw a distinction between dyspnœa or difficulty of breathing and exaggerated breathing or hyperpnœa, as seen in acidosis, and, again, the rapid, shallow breathing of pneumonia or collapse of the lung. Quite evident dyspnœa occurs in laryngeal diphtheria, asthma and bronchitis. Dyspnœa occurs in heart disease with a failing compensation, because the bronchial tubes become narrowed from congestion of the mucosa. In pneumonia the breathing is quiet and rapid. As a rule, when dyspnœa occurs in pneumonia it is a sign that the heart is failing. The late Samuel Gee, in a very learned and exhaustive lecture in 1899, reprinted in his "Lectures and Aphorisms," on pulmonary emphysema, concluded that pulmonary emphysema was caused by forced inspiration due to a feeling of dyspnœa consequent on obstruction of the air passages and that chronic progressive emphysema was the result of chronic bronchitis. He overlooked the physiological explanation that in chronic bronchitis there is a chronic negative pressure in the lungs and therefore an ever-present stimulus to inspiratory effort.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on September 30, 1921.

I shall now deal briefly with the carbon dioxide tension in the blood. Eighteen years ago Haldane and Priestley showed that the mean percentage of carbon dioxide in the alveolar air remained remarkably constant. As the carbon dioxide pressure in the alveolar air is the same as that in arterial blood, the interpretation was made that the activity of the respiratory centre was governed by carbon dioxide in the blood. Carbon dioxide supplies the stimulus for its own elimination and it is spoken of as the "respiratory hormone." Further investigation[1] showed that it was in virtue of its acidity that carbon dioxide acted and it is now accepted that the hydrogen ion is the respiratory hormone or sensitizer. One of the main functions of respiration, therefore, is the regulation of the reaction of arterial blood and "this is done with a delicacy that is almost inconceivable, calculation showing that a deficiency of one part by weight of ionized hydrogen in one billion parts of blood suspends completely the activity of the respiratory centre." The liver, by the formation of ammonia, and the kidney, by excreting acid phosphate of sodium, are also indispensable factors in maintaining the right reaction of the blood and tissues. The physiological and clinical effect of excess of carbonic acid or other acids in the blood is exaggerated breathing. The amplitude and, I think, also, the rate of the breathing is increased, but it is not accompanied by the distress and effort of true dyspnœa, unless there is associated bronchial obstruction. In its most defined form this character of breathing is seen in the acidosis of diabetes. I have seen it several times in acidosis in gastro-enteritis. It is also seen sometimes in association with shock. Yandell Henderson went so far as to say that hyperpnœa was an important factor in the production of shock; but his results are open to other interpretations.

We now come to oxygen-want or anoxæmia. It is curious to note that, seeing the necessity of oxygen to existence, under ordinary circumstances there is no special stimulus to oxygen-want and the stimulus that brings oxygen is another body, namely, carbon dioxide, performing another function. The explanation, no doubt is that under ordinary circumstances there is always more than enough oxygen available for the organism and that usually a shortage of oxygen in the system is associated with an excess of carbon dioxide. Another thing to remember is that in warm-blooded animals there is practically no storage capacity for oxygen and the organism depends from moment to moment for its oxygen supply on the air. There may be primary anoxæmia and secondary anoxæmia. Primary anoxæmia is simple deficiency of oxygen in the blood and may be due to bronchial obstruction, preventing air from getting into the lungs, or exudation in or collapse of the alveoli, preventing the blood and oxygen from coming together; it may also be due to defective circulation of blood through the lungs, as in heart disease, as well as to a deficiency of hæmoglobin, as in anæmia.

Secondary anoxæmia is more complicated and not so generally understood. It is due to alteration of the dissociation curve of oxyhæmoglobin. This concerns tissue respiration. Carbon dioxide in the tissues, by virtue of its acidity, aids the dissociation of oxyhæmoglobin and when its partial pressure in the blood is below normal, the hæmoglobin holds more tightly on to its oxygen. This may occur to such an extent that symptoms of anoxæmia may be present with blood of a normal red colour. It must be remembered that it is the oxygen in free solution and not the total available oxygen in the blood that is of immediate importance. About one-fiftieth part of oxygen in arterial blood is present in free solution, the remainder being combined with hæmoglobin in the easily dissociated compound oxyhæmoglobin. Forced voluntary breathing or rapid, shallow breathing, by washing out carbon dioxide from the blood, produces a relative alkalosis and can give rise to symptoms of anoxæmia.

The action of anoxæmia on the respiratory centre is peculiar. A shortage of oxygen stimulates the centre to some extent and there is increased breathing, which tends to become periodic in character. The increase in the breathing tends to quieten down as the carbon dioxide is driven off and an alkalosis produced. If the anoxæmia progresses, the respiratory centre becomes exhausted and rapid, shallow breathing occurs, which is itself a cause of anoxæmia; thus a vicious circle is set up, which rapidly ends in death if unrelieved.

Haldane maintains that periodic breathing is always a symptom of anoxæmia and is most easily produced by a moderate degree of oxygen-want. Oxygen-want acts on the centre much more promptly than variations of carbon dioxide pressure, because the latter is under the influence of buffer substances in the blood that do not influence oxygen. The result is that the centre, in the absence of carbon dioxide, alternately gets too much and too little stimulation. When the anoxæmia is great, the breathing becomes regular again, because the oxygen-want is never relieved. In Cheyne-Stokes breathing the periods end in apnœa of longer or shorter duration. Haldane's explanation of apnœa is a cessation of breathing owing to an absence of stimulation of the respiratory centre. Rapid, shallow breathing of itself may produce anoxæmia. Haldane, Meakin and Priestley have demonstrated this experimentally. In certain diseases of the lungs, such as pneumonia, collapse and infiltration of the alveoli, such as occurs in phosgene poisoning, cause apparently reflexly rapid, shallow breathing and it is in such cases that anoxæmia of a dangerous kind is likely to occur. The explanation given is, as pointed out by Professor Arthur Keith, that in the living body the lungs do not expand evenly and simultaneously at all parts, but open out part by part. In shallow breathing parts of the lung remain unopened or improperly expanded. The result is that, while carbon dioxide is normally excreted, the mixed arterial blood is deficient in oxygen. Anoxæmia is established, the breathing becomes more shallow from exhaustion of the centre and the oxygen-want progresses in the way already described. The necessity for oxygen inhalation to

[1] For the details, see Haldane's articles in the *British Medical Journal*, July 19, 1919, March 19, 1921.

break the vicious circle early is apparent; the administration of carbonic acid before it is too late may also afford relief, by increasing the amplitude of the breathing.

We have now recognized three main types of breathing in disease: (*i.*) dyspnœa, (*ii.*) hyperpnœa, (*iii.*) quick, shallow breathing.

Dyspnœa is associated with obstruction of the air way; there is also some retention of carbon dioxide and in severe cases deficiency of oxygen. Hyperpnœa is due to acidosis; there is no real difficulty of breathing, merely a panting endeavour to excrete acid. Rapid, shallow breathing is induced reflexly by collapse of lung tissue or the filling of the air cells with exudate. It promotes anoxæmia in the way indicated and, if unrelieved, leads to exhaustion of the respiratory centre, which increases the shallow breathing and thus establishes a vicious circle. The character of the breathing should always be observed at the bedside and, if interpreted properly, must give valuable information. In dyspnœa oxygen may be a valuable temporary measure while other relief is being efforded; but in the shallow breathing of anoxæmia oxygen is the main remedy and must be given promptly and continuously while the danger lasts.

I may now return to my cases. In young children the chest wall is soft, the respiratory muscles not strongly developed and the bronchial tubes very small. Thus, from a bronchitis which is not in itself serious, collapse of alveoli more or less extensive may occur. Rapid, shallow breathing follows, with the attendant danger of anoxæmia.

I might here record another case that came under my notice while this article was being written. On July 29 I was called to see in consultation K.K., a boy, aged eleven weeks. He had been suffering from a cough and moderate fever for three days, but during the last twenty-four hours he had become quiet, had refused his food and had seemed unable to cough. On examination I found that his face was pale and his lips blue. His temperature was 36.1° C.. His respirations were 50 to the minute and the pulse-rate about 160. The mother recorded that, during the last twenty-four hours, the breathing ceased for a few seconds several times and restarted with a definite panting effort. I diagnosed anoxæmia of a dangerous kind and offered to take the child with its mother direct to the Children's Hospital in my car, where it could get immediate and continuous oxygen. This was done. He was given a hot mustard bath and placed on continuous oxygen. The oxygen was given at first by means of a tube and funnel and during the night my house physician, Dr. Taylor, devised a bi-way glass tube and by means of two short lengths of rubber tubing the oxygen was fed directly into the nostrils. The child's colour and breathing improved and by next morning the temperature had risen to 37.2° C.. The child took its food and was able to cough effectively and cry fairly vigorously. Oxygen inhalation was continued with intermissions for forty-eight hours. The temperature rose to 40° C. on July 30, but soon came down. On August 2 the temperature was normal, the colour good and the child quite bright; but there was still a slight cough and the breathing was forty to the minute. On July 5 the baby was discharged quite well, except that there was still a cough. He is now quite well.

My reading of the case is that when I first saw the infant it was suffering from anoxæmia, a vicious circle had been established and it was only by regarding the condition as an acute medical emergency and securing for the baby immediate and continuous treatment by oxygen inhalation that its life was saved.

Oxygen-want occurs in many conditions; but the cases I have described seem to me to form a very definite clinical group, in that the anoxæmia supervenes on what would otherwise be a trifling ailment and that oxygen-want is the essence of the complaint.

The method of giving oxygen is important. Haldane has devised an apparatus for giving oxygen by which waste is prevented and the amount given easily regulated. This apparatus is not to be had in Sydney at present. The next best way to give oxygen is to feed it directly into the nose or mouth, interposing a wash bottle to gauge the amount of flow.

In conclusion, may I pay a tribute to the genius of J. S. Haldane. His work on anoxæmia is a fitting climax of a life of research and practical endeavour. His studies on the physiology of respiration, his work on carbon monoxide poisoning, mountain sickness, war-gas poisoning and anoxæmia stamp him as one of the great men of our profession.

Reports of Cases.

A BROAD LIGAMENT CYST SIMULATING A SUBPERITONEAL MYOMA OF THE UTERUS.

By **William T. Chenhall, M.D., F.R.C.S.,**
Honorary Surgeon, Royal Hospital for Women, Sydney.

I HAVE seen few cases more interesting and instructive from a diagnostic standpoint than the following:

J.D., aged 37 years, married, mother of three children, the youngest being five years of age. Menstruation began at 13 and conformed to an eight-twenty-eight type with strict regularity. The last menstrual period occurred three weeks prior to her being sent to me by Dr. George Curtis for an opinion. The duration was eight days and the amount of flow, for the first time, was excessive.

She was admitted to a private hospital on account of severe pain, which occurred suddenly in the mid-line above the pubis. The pain extended to both sides of the lower segment of the abdomen, then into the epigastrium and caused vomiting. A slight rise of temperature and an increased pulse-rate occurred. She then complained that she had suffered pain low down in the right side of the pelvis and abdomen during the past few years. Occasionally the pain extended down the right thigh. This pain had always become much more severe during each period of menstruation, although in all other respects the function was normal.

Urination had been for a long time frequent, both day and night, and was followed lately by great discomfort above the pubis when the bladder was emptied.

The patient had suffered two attacks of pelvic inflammation and this one, attended by Dr. Curtis, was very acute. He directed my attention to three important clinical points:

(*a*) Extreme tenderness during the inflammatory attack,

which he believed and I think justly believed arose from torsion of the pedicle. At the time of attendance he could not account for it.

(*b*) Fixation of the tumour. This was very striking and proved later to be due to adhesive inflammation to the utero-vesical fold, uterus and the omentum which bound it tightly in position.

(*c*) Epigastric pain. This either arose reflexly or possibly occurred from the drag on the omentum and intestine.

Dr. Curtis, in his memorandum to me, stated his belief that the uterus was "definitely enlarged and pulled over to the right side of the pelvis and lower abdomen." His chief concern was the existence or otherwise of pregnancy. What, then, if not pregnancy, caused the enlargement of the uterus? I confess that the condition puzzled me a great deal.

I quote briefly from my report: "The bladder is emptied voluntarily. There is a swelling, median, rounded, smooth, above the pubis. The vaginal outlet is slightly relaxed; the vaginal walls are healthy. There is a slight cervical laceration, healed. The uterus is enlarged, firm and retroverted; the anterior wall is occupied by a soft, semi-resistant, smooth, uniform, intra-mural myoma, extremely tender upon deep bimanual pressure." The tenderness I believed arose from some recent inflammation, whatever the cause.

Dr. Curtis had observed a rapid increase in size and in pressure effects and upon this evidence I suspected a rapidly developing myoma of the uterus, possibly sarcomatous or undergoing some degeneration and softening.

I could not find any evidence of pregnancy. Both fornices appeared perfectly normal. I believe it a good plan to write frankly what one finds. Thus alone can the finding be accurately checked at operation.

The Operation.

Immediate operation was decided upon. In Trendelenberg's position the abdominal aspect appeared typical of a fibro-myoma of the uterus.

Assured that the bladder was emptied a few minutes earlier, I opened the abdomen in the middle line.

Imagine our surprise to find a dense mass of omentum fixed below to the bladder and tightly stretched over a rounded tumour equal in size to a large orange. A line of demarcation between healthy and inflamed omentum was carefully tied off. The tumour then lay exposed and wedged in between the pubis in front and the intestines behind and forcing the uterus deeply down in the pelvis, where it could easily be defined. Both ovaries were also easily identified and elevated for examination. They were both perfectly normal.

The tumour was found to be cystic. The pedicle was formed by the Fallopian tube of the left side and a thin band of meso-salpinx below it. Further, it was formed by the stretched uterine end of the tube, which had become very tightly twisted. It was about 3.75 cm. in length.

The cyst occupied a site in the middle line, but extended rather to the right side, thus explaining Dr. Curtis's view that the uterus was pulled over to the right side. It was very tense, yet thin-walled. I quite easily secured the pedicle and removed the cyst, upon the circumference of which the elongated distal portion of the tube was found to be stretched and densely adherent.

As the uterus lay deep in the pelvis I deemed it expedient to suspend it by my own method, which is a modification of Gilliam's shortening.

The abdomen was closed in the usual four-layer method. The patient had made an excellent recovery.

Commentary.

The chief interest centres around the diagnosis and the pathology.

Diagnosis.

The commonest tumour in the female belly is an ovarian cyst. Usually its diagnosis is easy. The outline may be distinctly felt. It rises out of the pelvis and usually presents most prominently in a lateral position. It is tethered to the pelvis by its pedicle. One may, as a rule, lift it slightly out of the pelvis. It is distinct from the uterus, although closely associated with it. The uterus may be pressed forwards and downwards or backwards and downwards or towards one side. If the uterus be low down in the pelvis, it may be ascertained by careful vaginal examination that it is of normal shape and size and that it is distinct from the tumour. The passage of a uterine sound might in this case have indicated the direction of the uterus, but it would not have aided the diagnosis. Personally I never use a sound for diagnosis. An ovarian cyst fulfilling the above conditions may be easily diagnosed.

Usually, too, the diagnosis between an ovarian tumour and a uterine fibroid is easy. The distinguishing marks of the fibroid are its hardness, the absence of fluctuation, the elongation of the uterus, the oneness with the *cervix uteri* and its synchronous movement with it. But this cyst practically fulfilled these conditions.

Further, a fibroid may very closely simulate an ovarian tumour. Matthews Duncan directed attention to the feeling of fluid "as distinct from fluctuation." Moreover, a fibro-myoma may degenerate and in doing so create cavities filled with fluid.

Now, if such cavities be sufficiently large and close to the surface, they may fluctuate. Further, such degeneration seems apt to occur in sub-peritoneal myomata and in their presence the uterine cavity is not often lengthened. Thus in this case one had to deal with a semi-fluctuating tumour attached to the uterus, which, so far as one could decide, was not enlarged apart from the tumour.

Reviewing the whole case, I know no means by which one could have accurately distinguished the tumour from a myoma of the uterus until the abdomen was opened.

Pathology.

I shall deal very briefly with this aspect of the case. Any good text-book will state most of what is known concerning this complex problem. The simplest clinical plan is to regard it as a simple broad ligament cyst, since it is a simple cyst and is developed in the broad ligament. Let the pathologist and the embryologist continue their battle upon the source of origin until, finally, truth may prevail.

Many authors call it a parovarian cyst on the assumption that it is developed out of a vertical parovarian tubule. Doran claimed that it arises apart from the parovarium. Handley showed that some of these cysts are really dilated accessory Fallopian tubes. Professor Arthur Keith has shown that the "parovarian cyst" is formed by the ovarian fimbria of the Fallopian tube. In this case the tube, as already stated, extended a considerable distance across the surface of the cyst and the fimbriæ appeared to be very widely spread at the extremity of the tube.

The cyst, as already stated, was attached to a pedicle clearly formed by a very short portion of the uterine end of the tube, which had become stretched and twisted by the enlargement and rotation of the cyst.

Location of Cyst.

I confess great perplexity regarding this most remarkable feature of the case. How and why and when did it come to occupy this extremely unusual and very remarkable position? I have never before seen one similarly placed. Still more remarkable and perplexing was the rotation of the tumour and resultant twisting of its pedicle. The only reasonable assumption seems to be that it was an accidental occurrence of recent origin and that therewith arose the inflammatory changes which we found and for which Dr. Curtis first attended the patient.

The candidate for examination may feel justly thankful that such cases are extremely rare.

Reviews.

PATHOLOGY.

THE new edition of "Aids to Pathology," by Dr. Harry Campbell[1] is an excellent summary of modern pathology and may be of use to those who can bring themselves to read summaries. The work is up-to-date and is in accordance with the generally accepted teaching. The author adheres mainly to facts and only on a few occasions dis-

[1] "Aids to Pathology," by Harry Campbell, M.D., B.S. (Lond.), F.R.C.P.; Fourth Edition, 1920. London: Baillière, Tindall & Cox; Foolscap, pp. 248. Price: 4s. 6d. net.

cusses theory at any length; he gives a short account of general pathology, of the blood and its diseases, immunity, tumours, etc., and then deals with special pathology, following the customary usage in pathological text-books.

There are a few pen-and-ink illustrations of simple form, but there is little in the text or in the illustrations to help the reader to comprehend the pathological histology of the morbid conditions dealt with.

From diseases of the blood he omits to mention secondary anæmias and polycythæmia.

In the section on immunity he lays much stress on Wright's work in connexion with "opsonic index" and perhaps places more value on the estimation of the opsonic index as a guide to diagnosis and treatment than is warranted by the work of the last ten years.

He makes no mention of the advances made in protective inoculation during the war, though he devotes considerable space to the theories of immunity.

There is a good working classification of tumours and a discussion on the possible cause of malignant tumours, the author favouring the "irritation" theory and emphasizing the histolytic effect of malignant cells on normal tissues; he quotes Wright's statement that cancerous cachexia is due in nearly all cases to infection of the tumour by a streptococcus (*Micrococcus neoformans* of Doyen).

In common with other pathologists who have taken no notice of Thayer's investigations, he considers that a healthy radial artery can scarcely be differentiated from the surrounding tissues, which statement may lead the student to think that the radial arteries of all adult males are unhealthy.

The theory of wound shock has been upset by the work of Dale, Cannon and others during the war and secondary shock, occurring two or three hours after the wound, is now thought to be due to the absorption from the wound site of histamine, causing dilatation of the capillaries, with consequent partial emptying of the heart and sub-oxygenation of the tissues, since less blood flows through the lungs; at the same time, plasma escapes from the capillaries and helps to produce the condition of apparent bloodlessness with low blood pressure.

It is curious that he should ignore the fact that changes occur in the heart muscle in all acute infections and should lay so much stress on the occurrence of valvular lesions and infections of the serous coverings of the heart, rather than on the "carditis" which is the significant factor in so many cases of heart affection.

Under respiratory affections, the use of the term "ordinary phthisis" and no other name for chronic pulmonary tuberculosis is rather misleading and appears to have no justification from the pathological standpoint.

Affections of the endocrine glands and deficiency diseases are each dealt with as far as they satisfactorily can be in the short space available.

Campbell rightly takes the responsibility for a statement that "adenoids" is a dietetic disease and might be practically eradicated by the adoption of a rational system of feeding children.

The book is compact, well printed and arranged in chapters and paragraphs, which make it comparatively easy reading, considering how much information is crammed into so small a space.

Notes on Books.

A DAINTY CHRISTMAS PICTURE STORY.

HAD Miss May Gibbs taken part in the discussion on the physiological aspect of the White Australia question at the Brisbane Congress, the motions put to the meeting might have been defeated and a fresh set might have been carried in their stead. She would have been able to persuade all who have Australia's welfare at heart, that there is no need to increase the human population at all; that the wonderful country of beauty and sunshine belongs to the gum nuts and blossoms and to the lizards and 'possums and wallabies and kangaroos—but not to the bad Banksia men nor to the humans. But the big humans at Brisbane did not give a thought to all these small but important native folk of Australia, so Miss May Gibbs has been compelled to place on record some of their doings in another way. Last year she told us all about Cuddlepot and Snugglepie and the bad Banksia men; this year she has continued the history and has introduced "Little Obelia" at the bottom of the sea.[1] At the Big Bad City there are races; kangaroos with gum nuts "up" make good running, while the "enclosure" is as fascinating as anything in this strange land of fancy. There are boat races, cricket matches, football "scrums" and all kinds of fun. The dental parlour and the deep sea hospital are ideal institutions. Miss May Gibbs has given us another taste of her versatility, of her extraordinary imagination, of her genius in art and poetry and of her love of the things that live in the bush. "Little Obelia" is addressed to "all Little Boys and Girls and One Little Boy in Particular." The older boys and girls will enjoy it not less than the little ones. Order your copy early, before the edition is exhausted!

"WELLCOME" PHOTOGRAPHIC EXPOSURE CALCULATOR.

EACH year a new edition of the "'Wellcome' Photographic Exposure Calculator, Handbook and Diary" is offered to the lover of photography and each year this little pocket guide is made more useful. The new features of the 1922 edition is the inclusion in the table giving the exposure speed of plates and films, of the rate at which they develope. Much information is given in regard to tank development, which has been extended and amplified from previous issues. All who have become familiar with this diary and handbook, will recognize the need for purchasing it before the beginning of the year. To those who have struggled without this condensed aid to practical photography, we can heartily commend it.

INTERNATIONAL CONGRESS OF OTOLOGY.

THE INTERNATIONAL CONGRESS OF OTOLOGY will be held in Paris from July 19 to July 21, 1922. The President of the Congress is M. LE PROFESSEUR P. SÉBILEAU and the General Secretary M. LE DOCTEUR A. HAUTANT (28, Rue Marbeuf, Paris VIII[e]).

The British Committee of Organization has already received numerous applications for membership. Those who have not received a direct invitation are requested to communicate with the Honorary Secretary, MR. LIONEL COLLEDGE, F.R.C.S. (22, Queen Anne Street, London, W.1), and to include a subscription of 10s. as membership fee for the British Committee. The members of the British Committee will be informed from time to time of the details and arrangements of the Congress.

The Congress will embrace rhinology and laryngology, as well as otology. During the mornings the members will visit hospitals, clinics and museums and l'Institut Curie. The sessions of the Congress will be held in le Faculté de Médecine. Demonstrations of patients and specimens and epidiascope exhibitions will be given. A museum will be organized in connexion with the Congress.

Otologists in several countries are being invited to contribute a *rapport* on any subject selected by them. The *rapports* will be printed and circulated among members of the Congress. DRS. BUYS (Brussels) and GUIX (Utrecht) will deal with vestibular tests; PROFESSOR GRADENIGO (Naples) will speak on unsuspected syphilis of the ear; PROFESSOR SCHMIEGELOW (Copenhagen) will give a report on the diagnosis and treatment of abscess of the cerebellum and MR. G. J. JENKINS, O.B.E. (London), will read a paper on otitic meningitis.

The subscription of the Congress is Fr. 100.

The Organizing Branch of the British Empire has as its President DR. URBAN PRITCHARD and SIR SINCLAIR THOMSON as its Chairman. The two Honorary Secretaries are MR. LIONEL COLLEDGE, F.R.C.S. (London), and MR. J. S. FRASER, F.R.C.S. (Ed.), Edinburgh. SIR JAMES BARRETT, K.B.E., C.B., C.M.G., is the representative for Australasia.

[1] "Little Obelia and the Further Adventures of Ragged Blossom, Snugglepot and Cuddlepie," by May Gibbs; 1921. Sydney: Angus & Robertson, Limited; Crown 8vo., pp. 91, with two full-page coloured and 19 full-page half-tone and several smaller line illustrations. Price: 7s. 6d. net.

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Anoxæmia.

DR. W. F. LITCHFIELD'S interesting dissertation on a group of cases met with in infants during the first few months of life deserves careful attention and cautious analysis by every practitioner who may be called upon to recognize and treat this condition. In its essence the condition is characterized by periodic attacks of rapid, shallow breathing with some cyanosis. At times the respiratory failure is complete. According to Haldane the condition is produced by a diminution of the rate of diffusion of oxygen throughout the body. From the discussion it will be recognized that difference of opinion exists concerning the condition of the respiratory centre in these little patients. Nearly everyone admitted that the rhythmical respiratory excursions are maintained by a constant stimulation of the centre by carbon dioxide. Dr. Litchfield's contention that shortage of oxygen is a respiratory stimulus does not carry conviction. It is true that the respiratory centre becomes more sensitive to carbon dioxide when the oxygen tension is lowered. This increase in susceptibility, however, is relatively small and does not appear to play any considerable part in anoxæmia. While there is difficulty in understanding how a want of oxygen could excite a nervous centre, there appears to be a further objection to this suggestion in the fact that when carbon dioxide is washed out of the lungs and prevented from accumulating in the tissues, the respiratory centre does not manifest any tendency to send forth rhythmical messages. In anoxæmia it would appear that the amount of oxygen taken up by the blood from the alveolar air is probably diminished and the greater part of this oxygen is held tenaciously in solution in the plasma. A result of the diminished oxygen supply to the tissues would be a diminution of metabolic changes and a consequent reduction in the carbon dioxide output by the tissues. The body tends to maintain the normal hydrogen ion concentration of its fluids, but in these circumstances there would be a grave disturbance resulting from the partial cutting off of the supply of the acid products of metabolism, as well as a reduction in carbon dioxide. It is probable that other acid bodies besides carbon dioxide stimulate the respiratory centre at times, although the safety mechanism as far as breathing is concerned is certainly carbon dioxide. The infant using too little oxygen and excreting too little carbonic acid would tend to be attacked by bouts of deficient breathing, largely because the respiratory centre is not stimulated to respond. We are told that the inhalation of oxygen properly applied suffices to re-establish respiration in anoxæmia. It appears that this might find its explanation in the fact that the absorption of an excess of oxygen would compel even sluggish tissues to respond by the revival of metabolism, with a consequent liberation of carbon dioxide for the stimulation of the respiratory centre. Failure of oxygen inhalation to restore respiration would be due to the inability of the blood to carry the burning agent to the tissues and to give it off in situations where there is material to burn. It must be remembered that anoxæmia is a condition of early infancy. As soon as the child is old enough to attract oxygen by muscular exercise, this failure of the respiratory centre does not occur.

In considering the pathology of anoxæmia it is necessary to have regard to the fact that so far no acceptable explanation has been offered for the diminished supply of oxygen to the tissues. Haldane's work does not throw any light on the primary disturbance which leads to this diminution. It is probable that the bio-chemical fault lies in the blood and further that it is one of instability rather than a definite lesion. It is impossible, however, on the data available at present to arrive at any useful conclusion in this connexion.

In dealing with problems of this kind, it is necessary to translate the clinical manifestations into terms of physiology, by which is meant physics and chemistry. There is a danger if the physician endeavours to apply his remedies empirically without forming a working conception of the essential nature of the condition and of the manner in which the nor-

mal processes are failing. The explanation offered is hypothetical and may be erroneous. As soon as fresh chemical or physical data can be presented, it could be revised if necessary. It appears to us, however, to be plausible and to satisfy the facts as far as they are known.

THE DEVELOPMENT OF MITRAL STENOSIS.

In the year 1669 a description was published by John Mayou of the pathological changes in the heart of a young man who had died of cardiac failure. It was the first important contribution to the study of the disease now known as mitral stenosis. The description of the narrowing of the bicuspid valve and the hypertrophy of the cardiac musculature behind the obstruction was excellent and almost in complete accord with the observations of the present day. Raymond Vieussens, the great French anatomist, wrote a paper in 1715, the year of his death, in which he confirmed the findings of Mayou and drew attention to another feature, namely, the shortening of the *chordæ tendineæ*. Senac and other eighteenth century authors added further descriptions. By the early years of the nineteenth century the disease was familiar to every competent physician.

The invention of the stethoscope by Laennec led to the recognition of the characteristic presystolic mitral murmur, so that it soon became possible to diagnose the disease during the patient's lifetime. Gradually the relationship of mitral stenosis to the rheumatic group of diseases became evident. By the end of last century the main features of mitral stenosis were well known. The gradual onset of breathlessness and of paroxysmal orthopnœa, the frequency of hæmoptysis in this as compared with other cardiac affections, the tendency to bronchitis, to angina and to epigastric tenderness, the brassy cough and aphonia when the left recurrent laryngeal nerve becomes compressed by the dilated left auricle between the left pulmonary artery and the arch of the aorta, the liability to sudden death from cerebral embolism and the final stage when "compensation breaks down" and heart failure becomes marked—all these manifestations of the disease were universally recognized. It remained, however, for investigators of this century to throw a flood of light on the phenomena of cardiac failure in this disease. The inventions and researches of Einthoven, Wenckebach, Mackenzie, Lewis and others demonstrated the nature of the arrhythmia which replaces the regular heart action of mitral stenosis in its late phases. To Mackenzie and Lewis, in particular, the world owes its recognition of the significance of what is now known as auricular fibrillation. The treatment of this condition has been put on a satisfactory footing and has deprived this form of heart failure of much of its traditionary terror.

A lecture on mitral stenosis was recently delivered by Dr. Basil Parsons-Smith at the National Hospital for Diseases of the Heart.[1] By far the most interesting portion of the lecture is that dealing with the possibility of recognizing the development of the disease at an early stage. The narrowing of the valve aperture is known to be a gradual process extending over many years. A certain interval must elapse between the primary invasion of the valve curtains and the actual development of definite valvular obstruction. This interval, says the author, may occupy on an average three to six years. During this period the patient is conscious of mild cardiac embarrassment. Exercise renders him short of wind, he feels unduly run down and tired after mental as well as physical labour and palpitation and other subjective symptoms become manifest. The problem which confronts the physician is to correlate these symptoms with a pathological involvement of the heart. Mitral stenosis is recognizable enough when it has reached an advanced stage. But can it be detected while there is still a possibility of arresting or at least slowing the stenotic process? Dr. Parsons-Smith is confident that, with careful examination, this is possible, not merely in some cases, but in all. He presupposes that the patient looks healthy and is free of any apparent disease of extra-cardiac origin. His reserve of energy is beginning to show a restricted field of response. On auscultation the quality and pitch of the two heart sounds is found to be altered. The first is loud, prolonged and snapping and may or may not be followed by a systolic murmur. The second sound may be reduplicated at the apex or base. If the patient is made to undergo vigorous exercise, the tachycardia brings into operation the maximum effect of the early valvular damage and an evanescent presystolic mitral murmur may be heard. The recognition of the simple fact that the developing lesion passes through an early stage, during which its ill-effect upon the heart's mechanism is manifested only when an extra demand is made upon the circulation, is considered by the author to be of paramount importance. The patient must be examined in the standing and recumbent positions, when at rest and when performing a considerable amount of muscular work. The presystolic murmur may be heard for a few beats only after the exercise. If the stenotic process is more advanced, the murmur will persist for a longer interval following its production and its appearance will be made following very slight degrees of effort or change of posture.

Early recognition of sclerosis of the mitral valve allows the application of methods designed to delay the advance of the stenotic process. It is generally known that certain forms of treatment may delay the course of mitral stenosis and bring comfort to the patient. These preventive measures are simple. The patient should lead a quiet, systematic life, free from anxiety or worry or physical strain. His occupation should be light and congenial. Exercise is as desirable as fatigue is undesirable. Light outdoor exercises and hobbies, light digestible meals and discontinuance of smoking complete the régime. The administration of digitalis is only necessary when signs of weakness of the heart muscle appear.

The author makes scant reference to the part played by the myocardium in the disease. While

[1] *The Lancet*, May 28, 1921.

recognizing that the final clinical manifestations are dependent on the failure of the heart muscle, he adheres to the old doctrine that it is the stenosis alone which is the primary lesion and which leads to the ultimate failure of the myocardium. Surely it may now be generally accepted that the endocardial lesion is only one portion of the disease process. In acute rheumatic affections the heart muscle bears in the majority of cases the brunt of the attack. The fact that its recovery from attack may be more complete than that of the endocardium should not close our eyes to the attack it has undergone. Two facts serve to centre attention on the valves at the expense of the myocardium. One is the functional disturbance which results as the sclerosis of the valves advances. The other is the musical appeal of a presystolic murmur. The insult to the myocardial tissues should not be forgotten.

In these columns attention has been consistently called to the problem of cardio-vascular affections. It has been pointed out again and again that the enormous human and economic wastage occasioned by heart disease can be and should be prevented. The primary aim of medicine should be to attack heart disease by combating its causes. The rheumatic group of diseases and other acute infections are in the main the causes of heart affections and elimination of these conditions would reduce the incidence of the greatest cause of debility and premature death. A wise community will look to the health of its children and its youth rather than of those on whom grisly disease has already worked irreparable ravages. The activities of cardiologists have been directed largely to the study of advanced cardiac affections with a degree of success undreamt of a score of years ago. The early stages of heart disease are already claiming a multitude of students, of whom Dr. Parsons-Smith is one. A movement which would go further and aim to destroy the causes of impaired cardiac function, would earn the gratitude of the suffering community.

THE MANCHURIAN PLAGUE EPIDEMIC.

MANCHURIA is in process of recovering from her second great epidemic of pneumonic plague. The first epidemic occurred some ten years ago and was responsible for the death of 50,000 persons. The recent outbreak, as a result of more determined scientific endeavour, was much less disastrous, only 8,500 persons dying. As is usual in epidemics of plague, the outbreak assumed the bubonic form at its inception and appeared towards the end of summer. The first victims were a Russian woman and her two daughters, all of whom died. A son was also infected, but recovered. This family lived in the Transbaikal district of Siberia. During September and October, 1920, ten cases were discovered and nine of the ten patients died. One day at a town called Hailar the soldiers attacked the police and set free nine men who were suspected of suffering from plague. These men infected several of the local inhabitants and two of them reached the coalmines of Dalainor, 100 miles way. The disease had meanwhile displayed a metamorphosis from the bubonic into the septicæmic and thence into the pneumonic types. As a result, the infection spread widely and rapidly, the coolies at Dalainor receiving the full fury of the attack. It was not long before the disease had spread along the railway lines from Manchouli to Vladivostock and from Harbin to Changchun, a total distance of 1,200 miles. The details of the epidemic and the treatment adopted have been described by Dr. G. L. Tuck (Wu Lien Teh).[1] Passenger traffic was stopped between certain towns and limited to comparatively few persons between other towns. These few were made to undergo a period of quarantine for five days on arrival at their destination. Dr. Tuck is convinced that this procedure was one of the factors which prevented the epidemic from assuming the huge proportions of its predecessor.

The author records several interesting findings. As attempts to catch fleas in dark, dirty rooms with paper-covered windows were unavailing, guinea-pigs were allowed to roam about the rooms in the hope that the flea population would thus be collected. We are assured that this procedure proved a failure. Fly papers were also used as traps, with no success. Strangely enough, the author makes no mention of the measures directed against rats. The great danger of the plague is, he insists, infection in droplets of sputum coughed by patients suffering from plague pneumonia. Over 60,000 anti-plague gauze masks were made and distributed. Dr. Tuck thinks that cotton and gauze masks, if properly worn, are certainly efficacious against infection. It was noticed that a large number of coolies engaged for burial duties died. It was believed possible that the clothes which were removed from the corpses, might be the cause of dissemination of the disease. Cultures were made from pieces of cloth cut from in front and behind. Of twenty-two pieces, sixteen were found to harbour living bacilli. It is deduced that the clothing of plague victims is highly dangerous and should be burnt with the dead bodies. Plague houses were disinfected by fumigation with moist sulphur dioxide. The walls and floor were sprayed with water and crude sulphur was burnt in an earthen pot. The sputum was destroyed with slaked lime.

We must confess a certain amount of disappointment on reading the author's description of the measures used to combat the disease. No information is given of any attempt to destroy rodents. If a systematic and wise warfare was directed against the rat, Dr. Tuck is guilty of a strange error of omission. A campaign which is directed against the spread of a violent outbreak of pneumonic plague, may be already written down as a comparative failure. But severe criticism should be forborne when it is remembered that the people of Manchuria are not educated up to the requirements of preventive medicine. Evidently scientific work in Manchuria does not meet with popular approval. Dr. Tuck hopes that his people will take their experiences to heart and show a little more faith than hitherto in the efforts of their scientific men.

[1] *The Lancet*, October 22, 1921.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch was held at the B.M.A. Building, 30-34, Elizabeth Street, Sydney, on September 30, 1921, DR. T. H. FIASCHI, V.D., D.S.O., in the absence of the President, being elected to the chair.

Principles of Diagnosis.

DR. F. GUY GRIFFITHS read a paper on the "Principles of Diagnosis" (see page 617).

PROFESSOR A. E. MILLS stated that Dr. Griffiths had presented a large number of opportunities for discussing the subject matter of his paper. He, the speaker, agreed with the greater part of the paper, but he wished to point out that all the methods of diagnosis had not been exhausted. The majority of patients had no physical signs. If the practitioner were dependent on the result of a physical examination, he would find it impossible in many cases to determine what was the matter. Patients usually came to their doctor with certain complaints. It was necessary to ascertain what the symptoms were to endeavour to understand what the symptoms meant and to correlate the symptoms with some changes in the organs or tissues which might be assumed to exist. For example, a patient would come to a doctor and complain of indigestion. If the patient were closely questioned, it would be learnt that the symptom described by him was in reality a feeling of fullness. When this feeling was present there was necessarily some increased tension of the stomach, œsophagus or some other hollow viscus. By paying attention to the patient's own description of his suffering, the doctor usually obtained the right clue to the diagnosis.

Professor Mills consequently arrived at the conclusion that the most important factor in diagnosis was a careful analysis and a proper evaluation of the significance of the symptoms. In recommending this method he pointed out that it lent interest to cases which might at first sight appear to be quite uninteresting. He admitted, however, that there was no certain or royal road in diagnosis.

DR. HARVEY SUTTON, O.B.E., thought that it was difficult to separate the principles of diagnosis from the methods employed in arriving at a diagnosis. There were two factors which should be remembered. In the first place, the finding of a possible cause did not necessarily determine the nature of the disease. The ætiological factor might be present and yet the disease might be absent. In some cases it required a *tertium quid* to produce the manifestations of the disease. As an instance of this he spoke of the carriers of the bacterial causes of diseases. In the next place a disease might be general, although the most obvious signs and symptoms would suggest a local disease. Enteric fever was certainly not a local disease of the small intestine, as was previously thought. Sir James Mackenzie had called attention to the correlation between sensation in local areas of skin and lesions in hollow viscera. He had recognized, however, that the presence of a local lesion in any viscus might be a manifestation of a general condition.

Dr. Harvey Sutton thought that Dr. Guy Griffiths had outlined what might be regarded as the normal method of diagnosis. Competent physicians cultivated the art of diagnosis by the unconscious exercise of various methods of observation. The physiognomic diagnosis frequently provided a correct clue. Dr. Sutton referred to the evidence of the sense of smell in diagnosis and quoted instances in which this sense had proved most valuable. He thought that the senses of smell and of taste were neglected. The medical practitioner usually trained his tactile sense and his senses of vision and hearing to the exclusion of his other senses.

DR. T. H. FIASCHI, V.D., D.S.O., in thanking Dr. Griffiths, expressed the opinion that errors in diagnosis were often due to insufficient examination. At times the mind was not in a receptive condition and one would be liable to miss important signs or symptoms. He advised practitioners to devote plenty of time to examination and not to be in a hurry to arrive at a diagnosis.

In his reply, DR. GUY GRIFFITHS maintained that it might be almost impossible to make a diagnosis under certain conditions, even when a disease was well established. The symptoms were usually the result of the reaction of the patient to the cause of the disease and were not often direct evidence of the noxious element.

Anoxæmia.

DR. W. F. LITCHFIELD read a paper entitled "Observations on Anoxæmia, with an Account of an Acute Medical Emergency in Infants Due to Want of Oxygen" (see page 620).

DR. JOHN FLYNN congratulated Dr. Litchfield for bringing forward the subject of anoxæmia for discussion. He considered that, while conjecture had still to take the place of facts in connexion with certain respiratory abnormalities, they were on sure ground when they claimed that the type of breathing termed periodic or irregular was indicative of oxygen want and should be treated by inhalation of oxygen. To illuminate this the better, he contrasted periodic breathing with hyperpnœa which occurred after exercise. In hyperpnœa caused by muscular exercise there was a gradual transition from hyperpnœa to normal breathing. In addition to the tidal air there was the residual air, which was equivalent in volume to about six ordinary breaths. No matter what efforts were made, it was impossible to get rid of this residual air. It took some time to be greatly changed in composition. If it were not for this fact, there would be constant danger of sudden fainting whenever any momentary lack of oxygen occurred. In the next place, while there was a considerable capacity for the storage of carbon dioxide in the body, there was no such storage capacity for oxygen. In Cheyne-Stokes respiration, the respiratory centre was alternately too much and too little sensitized on account of this lack of storage of oxygen, while the carbon dioxide was the governor all the time.

Dr. Litchfield had stated that primary anoxæmia was simply deficiency of oxygen in the body and might be due to bronchial obstruction. Haldane used the term, which he had introduced, to mean not deficiency of oxygen in the body, but to express a diminished rate of supply of oxygen to the tissues. To understand the distinction it was necessary to bear in mind that it was the oxygen in free solution and not the total amount of available oxygen in the blood that was of immediate importance. There was a marked alteration in the dissociation curve of oxy-hæmoglobin proportional to the partial pressure of the carbon dioxide. This signified that the carbon dioxide was the real governor. Dr. Litchfield had stated that respiration in health and in disease was under three main influences, namely, reflex nervous action, carbon dioxide tension in the arterial blood and oxygen supply. It might have been permissible to have stated the matter in fashion before Haldane's work had been made known. At present it would be truer to state that respiration in health and disease was directly under one influence and one influence alone, the hydrogen ion concentration of the blood, as expressed by the tension or percentage of carbon dioxide in the alveolar air. The nerve cells in the respiratory centre possessed a specific sensibility for the increase or diminution of carbon dioxide.

In the next place, Dr. Flynn said that want of oxygen did not of itself excite the respiratory centre. Indirectly, however, it did so, inasmuch as it lowered the threshold at which carbon dioxide excited it. Similarly, the vagus influence acted indirectly. The absence of vagus influence heightened the threshold, so that a larger percentage of carbon dioxide was required. He instanced in this connexion a further proof that carbon dioxide was the real governor by referring to the fact that excessive ventilation of the lungs did not produce apnœa if the percentage of carbon dioxide in the alveolar air were prevented from falling by the administration of carbon dioxide. The function of the vagus was merely to keep the respiratory centre informed of the distension or collapse of the lung, so that useless efforts might be spared.

Anoxæmia or diminished rate of supply of oxygen to the tissues might be due to various causes. It might be due to defective saturation of the blood with oxygen, as in mountain sickness, or it might be due to slowing of the

circulation, such as occurred in heart disease, or it might be due to defective proportion of oxygen.

In conclusion, Dr. Flynn ventured to quote a wise saying of Sir Clifford Allbutt: "To go with physiology as far as we can is not only to tread so far on relatively firm ground, but also to provide a discipline and a precise terminology."

PROFESSOR A. E. MILLS congratulated Dr. Litchfield on his thoughtful paper. Dr. Litchfield had recognized the clinical manifestations which were so often overlooked. His contribution was praiseworthy, even if Dr. Litchfield had been a little inexact in his terminology. He was inclined to agree with Dr. Flynn. In the first place, oxygen was necessary for the due performance of the function of all the tissues of the body. He, Professor Mills, held that the real stimulus to the respiratory centre was undoubtedly carbon dioxide. It was difficult to understand how a want of oxygen could result in the stimulation of a centre. The oxygen tension could be decreased to a considerable degree as long as the oxygen was diffused gradually, without any deficiency becoming manifest. When the respiration failed, there would be a diminution in the diffusion of oxygen.

In discussing the mechanism of respiratory failure in anoxæmia, he suggested that there might be other metabolic products exerting their influence in addition to the diminution of the tension of the oxygen. It was known that the products of incomplete combustion in diabetes and in certain heart diseases did give rise to acid poisoning. It seemed to him that the crux of the whole question lay in the possible presence of substances of this nature exerting an influence on the respiratory centre in anoxæmia.

Dr. Litchfield would have them believe that cyanosis was not due to want of oxygen. To what could it be due? In Dr. Litchfield's case of bronchitis with cyanosis little had been found in the lungs. They would have to ask whether the blood had really been saturated with oxygen after oxygen had been inhaled. Dr. Flynn had dealt with the difference between oxygen taken up by the hæmoglobin of the blood and oxygen held in solution in the plasma. If the blood could give off oxygen after inhalation, it was obvious that the child ought to have got better. As long as the breathing was efficient and the supply of oxygen adequate, some factor, probably of the nature of a poisonous substance, must have been acting to prevent the oxygen from functionating in the tissues. Professor Mills held that in these cases it would be better to stimulate the respiratory centre with carbon dioxide and oxygen than to rely on oxygen alone.

He agreed with Dr. Flynn in regard to the part played by the vagus nerves in oxygen want. Provided that spasm of the larynx was prevented, an animal did not die when both vagi were cut. Afferent stimuli under these conditions were, of course, eliminated. Even if all the nerves were cut, the animal went on breathing. He claimed that the inspiratory effort could be easily understood if his explanation were accepted.

Turning to the question of emphysema, he maintained that the dyspnœa was expiratory and not inspiratory. There was no difficulty for the air to get into the lungs, but there was a grave difficulty impeding its exit. It had been pointed out that hyperpnœa was an important factor in the production of shock. Professor Mills recognized that this claim was a just one. In conclusion, he again thanked Dr. Litchfield for giving them a paper which made them think.

DR. HARVEY SUTTON, O.B.E., said that he was not yet convinced of the accuracy of the statements of the previous speakers. Criticism of Haldane's convincing evidence had to be accepted with caution. In the first place, he had found that carbon dioxide in the alveolar air was a deciding factor in the ventilation of the lungs. Reference had been made to an experiment in which the breath had been held for three and a half minutes. In the condition of apnœa from forced respiratory ventilation a person would have no inclination to breathe from three to four minutes. Dr. Sutton referred to tests carried out with the spirometer. The subject was required to blow into the apparatus until the mercury was 30 mm. or 40 mm. and then to see how long he could maintain the mercury at this level. By artificial ventilation, by which was meant repeated, rapid, forced breathing, the period of time during which the mercury could be held at this level was increased up to 50, 60 or even 75 seconds. It was quite certain that carbon dioxide was a stimulus to the respiratory centre. In these circumstances it appeared to him logical to give carbon dioxide when it was necessary to treat a condition of apnœa, such as when a patient stopped breathing under anæsthesia or was suffering from Cheyne-Stokes breathing. Certain types of cases were encountered in which the term anoxæmia appeared to him justified, first as an indication of the necessary treatment and, secondly, as giving a clue to the essential causal failure. In anoxæmia the administration of oxygen undoubtedly was of use, but this did not necessarily imply that there was a want of oxygen, but that the blood was not giving off a sufficient quantity of oxygen to the tissues. It appeared to him that other factors were involved which acted on the respiratory centre. He could not understand why it was held that diminished oxygen pressure should necessarily bring about a failure of a centre suddenly and completely. Would it not be more reasonable to suppose that the effect would be in a condition when it needed stimulation from a further substance for its continued activity?

DR. P. FIASCHI, O.B.E., spoke of the experience with Meltzer's or the insufflation method of anæsthesia. It was possible by this method to make the patient breathe stertorously when failure of the respiratory centre threatened. This was due to an accumulation of the carbon dioxide. In Europe a few years ago anæsthetists always had a cylinder of carbon dioxide alongside the oxygen cylinder in the operating theatre. It was found that the insufflation should not be continuous, but should be interrupted four, five or six times a minutes. Dr. Fiaschi preferred the hand pump to mechanical methods, on account of the single stroke. He held that carbonic acid in the blood was the main factor in the stimulation of the respiratory centre.

DR. C. E. CORLETTE thought that their ideas concerning this subject were somewhat confused. It was difficult to find out exactly what they had to think. Their knowledge at present on the whole subject of anoxæmia was very imperfect. Professor Mills had suggested that other metabolic products might be formed and after the carbon dioxide had been washed out, these products might account for the stimulation of the respiratory centre. He referred to the work of Grant and Goldman, published in the *American Journal of Physiology* in 1920, on long-continued, forced breathing. They described tetaniform convulsions as a result of continued forced beathing and called these convulsions tetany. They adduced evidence of the appearance of a hypertonus of the respiratory muscle. Dr. Corlette also mentioned the discovery of guanidin compounds in the muscles and suggested that other toxic products might accumulate in the blood and make the tissues more responsive to stimuli. Dr. Harvey Sutton had drawn attention to the production of apnœa by forced breathing and had stated that breathing could be interrupted in this manner for a period of from three to four minutes. Dr. Corlette thought that Vernon had the record, having held his breath for something over eight minutes. In conclusion, he suggested that the best method of stimulating the respiratory centre might be the alternating application of oxygen and air.

DR. F. GUY GRIFFITHS found that the subject of tissue respiration was a difficult and a complicated one. Two great Englishmen who had worked at this subject, were Haldane and Leonard Hill. They had not been wise in naming the condition anoxæmia. The *ox* in the word did not mean oxygen. It was derived from ὀξύς, which meant sharp or acid. Had they called the condition anoxygen-æmia, they would have expressed what they meant. Oxæmia might mean "acid blood," *i.e.*, acid intoxication. Haldane had worked with Leonard Hill on the subject of ventilation and had claimed that closeness arose not from a lack of oxygen, nor from an excess of carbon dioxide, nor even from exhaled impurities, but solely from moisture and stillness. He had asserted definitely that no toxic substance was given off in respiration, because he had been unable to detect any. He justified the exclusion of a toxic substance on the claim that very minute amounts of carbon dioxide could be detected in air, something like one part per billion.

Dr. Griffiths expressed the opinion that Haldane's work was vitiated because he was a vitalist. He had built up his arguments on vital rather than on mechanical prin-

ciples. Dr. Griffiths, however, recognized the valuable outcome of Haldane's work in clinical practice.

In his reply, DR. LITCHFIELD stated that Haldane's article required very careful study. He had read it several times and had discovered fresh points of interest during each reading. He held that Haldane had enabled them to save some lives. The term anoxæmia might not be a good one, but so far no better had been suggested. He had tried in his paper to differentiate between primary and secondary anoxæmia. Reference had been made in the discussion to the reflex action on the respiratory centre. Haldane had admitted the importance of the reflex. He had been asked how the respiratory centre could be stimulated by oxygen want. Haldane had made this matter quite clear and had shown that the stimulation took place in the absence of carbon dioxide as a result of oxygen want. In heart disease there was cyanosis and heaving breathing, while in air hunger there was excessive breathing, but no true dyspnœa. Dr. Litchfield claimed that in cases of heart failure there was a negative pressure of oxygen in the lungs, owing to congestion of the bronchial mucosa. He explained the failure in the case recorded on the part of the patient to respond to oxygen by assuming that the oxygen had not been efficiently administered. The gas had merely been directed through the tube toward the patient's mouth. He agreed with Professor Mills concerning the advisability of administering carbon dioxide with oxygen in cases of anoxæmia. In regard to Dr. Corlette's reference to the tetany of forced breathing he suggested that this led to an alkalosis which was supposed to be a cause of tetany and which by inducing oxygen want might cause cerebral anoxia and as a result convulsions.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the Victorian Branch of the British Medical Association:

ADMANS, ALMA DOROTHY, M.B., B.S., 1921 (Univ. Melb.), Brunswick.
KENNEDY, GERALD JOSEPH, M.B., B.S., 1921 (Univ. Melb.), Mooroopna.
MATHEW, ALEXANDER CAIGIEVAR, M.B., B.S., 1921 (Univ. Melb.), Geelong Hospital.
MIDDLETON, LESLIE JAMES, M.B., B.S., 1921 (Univ. Melb.), Melbourne Hospital.
PARK, JAMES, M.B., B.S., B.A.O., 1915 (Univ. Belfast), Tatura.
UTBER, WALTER BIRD, M.B. *et* Ch.B., 1914 (Univ. Melb.), Rushworth.

Naval and Military.

APPOINTMENTS.

THE following appointments, etc., have been announced in the *Commonwealth of Australia Gazette*, Nos. 94 and 95, of December 8 and 15, 1921:

Permanent Naval Forces of the Commonwealth.

SEA-GOING FORCES.

SURGEON-LIEUTENANT HORACE PALMER MARGETTS, O.B.E., is promoted to the rank of Surgeon-Lieutenant Commander, dated 5th February, 1921.

Australian Military Forces.

GRANT OF SUBSTANTIVE RANK ON THE RESERVE OF OFFICERS.

The Governor-General in Council has approved of the undermentioned being granted substantive rank on the Reserve of Officers equivalent to that held by them in the Australian Imperial Force at the date of the signing of the Armistice, dated 1st January, 1921, except where otherwise stated:

FIRST MILITARY DISTRICT.

To be Captain.—HONORARY CAPTAIN LOUIS MICHAEL PIGOTT, Australian Army Medical Corps Reserve.

SECOND MILITARY DISTRICT.

To be Majors—CAPTAINS VINCENT WELLESLEY SAVAGE, D.S.O., and HARRY MANDER NORTH.

THIRD MILITARY DISTRICT.

To be Majors—HAROLD BRUCE SUTTOR and ERNEST EDERBLOM KING, CAPTAIN JOHN VICTOR HUME GUEST, Reserve of Officers, and HONORARY CAPTAIN JOSEPH STANLEY REED, Australian Army Medical Corps Reserve.

SIXTH MILITARY DISTRICT.

To be Major.—HONORARY MAJOR BERTRAM INGRAM, Australian Army Medical Corps Reserve.

To be Captains.—HONORARY CAPTAINS REGINALD M. WEBSTER, GEORGE MUSGRAVE PARKER, DAVID SMITH HENDERSON, CLEMENT GORDON HARRIS and DONALD PEDRO LUCADOU-WELLS, Australian Army Medical Corps Reserve.

TRANSFERS FROM OLD ORGANIZATION.

The following officers are transferred from the Australian Army Medical Corps (Old Organization), with effect as from 31st March, 1921, unless otherwise stated:

FIRST MILITARY DISTRICT.

Australian Army Medical Corps.—COLONEL A. H. MARKS, C.B.E., D.S.O., Australian Army Medical Corps, and to be supernumerary to the establishment of Colonels, with pay and allowances of Lieutenant-Colonel, from 31st March, 1921, to 31st July, 1921; COLONEL A. G. BUTLER, D.S.O., Australian Army Medical Corps, and to be supernumerary to the establishment of Colonels, with pay and allowances of Lieutenant-Colonel, from 31st March, 1921, to 31st August, 1921; MAJOR C. J. WEEDON, MAJOR (HONORARY LIEUTENANT-COLONEL) F. C. WOOSTER, D.S.O., and MAJOR H. S. MCLELLAND, Australian Army Medical Corps; CAPTAIN (HONORARY MAJOR) W. R. KELLY, from the Australian Army Medical Corps Reserve, and to be Major, 1st September, 1921; CAPTAIN G. A. C. DOUGLAS, CAPTAIN (HONORARY MAJOR) J. S. SMYTH and CAPTAIN C. M. LILLEY, Australian Army Medical Corps; CAPTAIN (provisionally and temporarily) E. CULPIN and CAPTAIN (temporarily) R. WALLACE, Australian Army Medical Corps, and to be Captains; CAPTAINS E. S. MEYERS and W. J. FEARNLEY, Australian Army Medical Corps; CAPTAIN (provisionally and temporarily) E. J. SAVAGE, Australian Army Medical Corps, and to be Captain (provisionally); CAPTAIN (provisionally) D. E. A. BUCHANAN, CAPTAIN (HONORARY MAJOR) J. G. AVERY, CAPTAIN (provisionally) A. D. MCKENZIE, CAPTAINS E. G. THOMSON, M.C., P. E. VOSS, M.C., C. SHELLSHEAR, R. M. ALLEN, M.C., J. V. J. DUHIG, B. L. HART and W. L. MILLETT, Australian Army Medical Corps; HONORARY CAPTAIN N. C. TALBOT, M.C., from the Australian Army Medical Corps Reserve, Second Military District, and to be Captain, 1st September, 1921; CAPTAIN WALTER HENRY STEEL, from the Reserve of Officers, Third Military District, and to be Captain (provisionally), 1st September, 1921; LIEUTENANT (provisionally) I. V. YOFFA, Australian Army Medical Corps; BURNETT LESLIE WOODBURN CLARKE, M.B., B.S., to be Lieutenant (provisionally), 1st September, 1921.

SIXTH MILITARY DISTRICT.

Australian Army Medical Corps.—COLONEL W. W. GIBLIN, C.D., V.D., to be supernumerary to the establishment of Colonels, with pay and allowances of Lieutenant-Colonel, from 31st March, 1921, to 31st July, 1921; MAJORS (HONORARY LIEUTENANT-COLONELS) J. A. NEWELL and H. N. BUTLER, D.S.O., MAJOR H. Z. STEPHENS, CAPTAIN L. GOLLAN, CAPTAIN AND BREVET-MAJOR (HONORARY LIEUTENANT-COLONEL) W. L. CROWTHER, D.S.O., CAPTAINS C. G. THOMPSON, C. N. ATKINS and W. SMELLIE, CAPTAIN (HONORARY MAJOR) J. SPRENT, M.C., CAPTAINS S. G. GIBSON, M.C., W. I. CLARK, M.C., and J. D. NORRIS, CAPTAINS (provisionally) L. O. MACNAMARA and E. A. ELLIOTT, 1st June, 1921. *To be Majors*—CAPTAIN AND BREVET-MAJOR (HONORARY LIEUTENANT-COLONEL) W. L. CROWTHER, D.S.O., and CAPTAIN C. G. THOMPSON, 1st September, 1921. *To be Major (provisionally)*—CAPTAIN C. N. ATKINS, 1st September, 1921. *To be Brevet-Lieutenant-Colonel*—MAJOR (HONORARY LIEUTENANT-COLONEL) W. L. CROWTHER, D.S.O., 1st September, 1921.

CHANGES, ETC..

The Governor-General in Council has approved of the following changes being made in connexion with the Australian Military Forces:

FIRST MILITARY DISTRICT.

Staff.—MAJOR W. R. KELLY, Australian Army Medical Corps, is appointed Assistant Director of Hygiene, 1st September, 1921.

Australian Army Medical Corps.—COLONEL D. G. CROLL, C.B.E., and LIEUTENANT-COLONEL T. G. ROSS, D.S.O., are transferred to the Unattached List, 31st August, 1921; CAPTAINS A. M. LANGAN and J. J. POWER, D.S.O., are transferred to the Reserve of Officers, and to be Majors, 31st March, 1921, and 9th July, 1921, respectively; CAPTAINS M. S. PATTERSON and L. P. WINTERBOTHAM are transferred to the Reserve of Officers, 31st March, 1921; CAPTAINS R. S. BERRY and G. A. RENWICK are transferred to the Reserve of Officers, 31st October, 1921; CAPTAIN (provisionally) H. EVANS is transferred to the Australian Army Medical Corps Reserve and to be Honorary Captain, 1st April, 1921; CAPTAIN (provisionally and temporarily) M. L. CAMERON and CAPTAIN (provisionally) J. H. MACARTHUR are transferred to the Australian Army Medical Corps Reserve and to Honorary Captains, 1st November, 1921; CAPTAIN A. P. ROSS is placed on the Retired List, with permission to retain his rank and wear the prescribed uniform, 30th November, 1921; LIEUTENANT (provisionally) W. L. CALOV is transferred to the Australian Army Medical Corps Reserve and to be Honorary Captain, 9th October, 1921.

SECOND MILITARY DISTRICT.

Australian Army Medical Corps.—The resignation of LIEUTENANT A. J. METCALFE of his provisional appointment is accepted, 1st December, 1921.

Australian Army Medical Corps Reserve.—HONORARY CAPTAIN N. C. TALBOT, M.C., is appointed to the Australian Army Medical Corps, First Military District, and to be Captain, 1st September, 1921; the resignation of HONORARY CAPTAIN J. P. HASTINGS of his appointment is accepted, 6th September, 1921.

THIRD MILITARY DISTRICT.

Australian Army Medical Corps Reserve.—HONORARY CAPTAIN W. J. DENEHY is granted the temporary rank and pay of Major whilst employed at No. 11 Australian General Hospital, from 6th October, 1920, to 30th June, 1921.

FOURTH MILITARY DISTRICT.

The undermentioned officers are placed on the Retired List, with the ranks stated, and with permission to wear the prescribed uniform, 1st December, 1921: With the rank of Major—HONORARY MAJOR T. CHERRY; with the honorary rank of Major—CAPTAIN J. W. M. BUICK.

The undermentioned officers are retired under the provisions of Australian Military Regulation 158 (*h*), 1st December, 1921: HONORARY CAPTAINS L. R. STEELE, R. J. LOOSLI and T. B. RYAN.

Australian Army Medical Corps.—The notification respecting the confirmation of the provisional appointment of CAPTAIN W. J. CLOSE, which appeared in Executive Minute, No. 280/1915 (promulgated in *Commonwealth of Australia Gazette*, No. 32, of 24th February, 1915) is cancelled.

Australian Army Medical Corps Reserve.—The resignation of HONORARY CAPTAIN S. L. CORRY of his appointment is accepted, 1st December, 1921.

SIXTH MILITARY DISTRICT.

Staff.—MAJOR C. G. THOMPSON, Australian Army Medical Corps, is appointed Deputy Assistant Director of Medical Services (Fifth Divisional Head-Quarters), 1st September, 1921.

Staff.—MAJOR (provisionally) C. N. ATKINS, Australian Army Medical Corps, is appointed Assistant Director of Hygiene, 1st September, 1921.

Australian Army Medical Corps.—The notification respecting the transfer to the Reserve of Officers of MAJOR (HONORARY LIEUTENANT-COLONEL) J. A. NEWELL, which appeared in Executive Minute No. 117/1921, promulgated in *Commonwealth of Australia Gazette*, No. 27, of 24th March, 1921, is cancelled.

Public Health.

THE PLAGUE OUTBREAK.

THE BULLETIN, headed "Plague in Australia," has been issued by the Federal Department of Health twice weekly up to the commencement of December. In future it will be published once a week. Numbers 21 and 22 contain a considerable amount of information concerning the measures taken by the Federal authority for the purpose of complying with the International Sanitary Convention. In addition, a full statement has been sent to all overseas countries detailing the measures taken by the Department of Health to prevent the transmission of plague from the infected areas of the Commonwealth. These measures include those adopted in regard to vessels and merchandise leaving infected ports, the precautions taken in regard to vessels arriving at Australian ports from infected ports and the regulations based on the resolutions adopted at the plague conference, which have now been gazetted. The general information further amplifies the different instructions given to the quarantine officers and others at infected ports and at certain border stations for the purpose of giving effect to the resolutions of the conference of November 14, 1921.

Fleas on Animals.

Investigations have recently been carried out at the Australian Institute of Tropical Medicine with the view to the determination of the identification of the fleas infesting various animals. Two black rats were found to be harbouring both *Xenopsylla cheopis* and *Ctenocephalus felis*. *Xenopsylla cheopis* in small numbers were recovered from guinea-pigs and white rats. Four rabbits in captivity were found to be harbouring large numbers of *Ctenocephalus felis*. One plague flea was found on a rabbit. No rat fleas (*Xenopsylla cheopis*) were discovered on cats or dogs.

A warehouse in Sussex Street, Sydney, was fumigated with hydrocyanic acid vapour. The amount of potassium cyanide used per cubic metre was ten grammes. It is stated that the total capacity of the building was 2,050 cubic metres. The building, which contained large quantities of produce, was kept sealed for twenty-one hours. After it had been opened, weevils and beetles were found crawling on the bags.

Plague in Human Beings.

During the fortnight ended December 10, 1921, six further infections in human patients were notified in Brisbane. In addition, three patients suspected of plague infection were also admitted to hospital. The number of cases from August 23 to December 10, 1921, in Brisbane was 35 and the number of deaths 22. In addition to these infections, two infections were notified on December 12 and 13 respectively.

The position at Townsville is summarized as follows: There were four further cases in human beings during the fortnight, bringing the total number up to 30. The total number of deaths was 19. No case was reported between December 10 and December 15, 1921.

At Cairns two definite infections were reported and one suspected case during the fortnight. In addition, the infection in a suspected case was declared to be plague. The total number of undoubted cases up to December 10 was eighteen and of these ten proved fatal.

The first patient in Sydney died on December 2, 1921. A second infection in man was notified on December 5, 1921. The patient had been employed in rabbit fumigation operations.

Plague in Rodents, etc..

During the fortnight ended December 10, 1921, eleven infected rodents were discovered in Brisbane out of 2,654 examined. No infected rats were found at Townsville during the same period, nor were any of the rats captured in Cairns infected. One infected rat was found at Ingham.

Many rodents were examined in other Queensland towns and in New South Wales, Western Australia, South Australia and Tasmania. None was found to be infected.

New South Wales Declared Infected.

A proclamation has been published in the *Commonwealth of Australia Gazette*, No. 94, of December 8, 1921, declaring New South Wales to be a place infected with plague.

Correspondence.

TREATMENT OF "RED-BACK" SPIDER BITE.

SIR: The common red-back spider (*Latrodectus hasselti*) is found everywhere here and cases of red-back spider "bite" are very numerous. Such a bite usually means a week of continuous aching and pain, accompanied by drenching sweats.

The ordinary treatment by applications of carbolic acid, lysol, iodine, etc., are unsatisfactory and I resolved to treat the next one like a snake bite. Last week I saw a girl who had been "bitten" about half an hour previously on the left thigh. The spider was caught and identified. Pains were already shooting up and down the thigh. The "bite" was incised under local anæsthesia and strong potassium permanganate solution rubbed in. Tne pain ceased immediately and did not recur, nor was there any sweating.

Yours, etc.,

J. W. SUTHERLAND.

Narromine, New South Wales,
December 18, 1921.

Obituary.

JOHN COOKE.

IT is with regret that we learn that Dr. John Cooke, of Prahran, Victoria, met with his death as a result of an accident on endeavouring to enter a moving train on December 15, 1921.

Books Received.

THE EFFECT OF CERTAIN AGENTS ON THE DEVELOPMENT OF SOME MOULDS, by K. G. Bitting, M.S., Bacteriologist, Glass Container Association of America; 1920. Washington, D.C: National Capital Press, Incorporated; Crown 4to., pp. 177, with 62 plates.

Medical Appointments.

DR. R. W. TELFORD (B.M.A.) has been appointed Health Officer in the Department of Public Health, Queensland.

* * * *

DRS. G. BROWN (B.M.A.), P. S. MESSENT (B.M.A.) and G. R. WEST (B.M.A.) have been appointed Honorary Anæsthetists at the Adelaide Hospital.

* * * *

DR. H. RISCHBIETH (B.M.A.) has been appointed Medical Officer at the Night Clinic of the Adelaide Hospital.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii..

COMMONWEALTH PUBLIC SERVICE: Six Medical Officers and Six Junior Technical Assistants at the Serum Laboratory, Department of Health, Melbourne.

DEPARTMENT OF HEALTH OF THE COMMONWEALTH: Four Travelling Fellowships.

THE MEDICAL JOURNAL OF AUSTRALIA: Assistant Editor.

WALTER AND ELIZA HALL INSTITUTE OF RESEARCH IN PATHOLOGY AND MEDICINE, MELBOURNE: Whole-Time First Assistant.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C..

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney	Australian Natives' Association Ashfield and District Friendly Societies' Dispensary Balmain United Friendly Societies' Dispensary Friendly Society Lodges at Casino Leichhardt and Petersham Dispensary Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney Marrickville United Friendly Societies' Dispensary North Sydney United Friendly Societies People's Prudential Benefit Society Phœnix Mutual Provident Society
VICTORIA: Honorary Secretary, Medical Society Hall, East Melbourne	All Institutes or Medical Dispensaries Australian Prudential Association Proprietary, Limited Manchester Unity Independent Order of Oddfellows Mutual National Provident Club National Provident Association
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane	Brisbane United Friendly Society Institute Stannary Hills Hospital
SOUTH AUSTRALIA: Honorary Secretary, 3, North Terrace, Adelaide	Contract Practice Appointments at Renmark Contract Practice Appointments in South Australia
WESTERN AUSTRALIA: Honorary Secretary, 6, Bank of New South Wales Chambers, St. George's Terrace, Perth	All Contract Practice Appointments in Western Australia
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington	Friendly Society Lodges, Wellington, New Zealand

Diary for the Month.

JAN. 3.—New South Wales Branch, B.M.A.: Council (Quarterly).
JAN. 10.—New South Wales Branch, B.M.A.: Ethics Committee.
JAN. 11.—Western Australian Branch, B.M.A.: Council.
JAN. 12.—Victorian Branch, B.M.A.: Council.
JAN. 13.—Queensland Branch, B.M.A.: Council.
JAN. 13.—South Australian Branch, B.M.A.: Council.
JAN. 17.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JAN. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee; Organization and Science Committee.
JAN. 25.—Victorian Branch, B.M.A.: Council.
JAN. 26.—Brisbane Hospital for Sick Children: Clinical Meeting.
JAN. 27.—Queensland Branch, B.M.A.: Council.
FEB. 3.—Queensland Branch, B.M.A.: Branch.
FEB. 8.—Federal Committee of the British Medical Association in Australia.
FEB. 8.—Western Australian Branch, B.M.A.: Council.
FEB. 9.—Victorian Branch, B.M.A.: Council.
FEB. 9.—Brisbane Hospital Clinical Society.
FEB. 10.—Tasmanian Branch, B.M.A.: Annual Meeting.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, B.M.A. Building, 30-34, Elizabeth Street, Sydney. (Telephone: B. 4635.)

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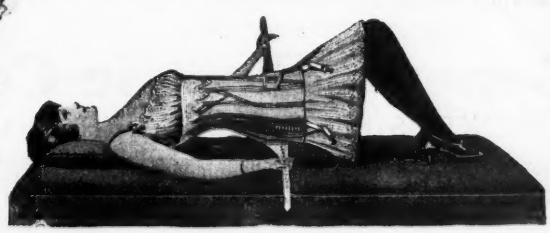

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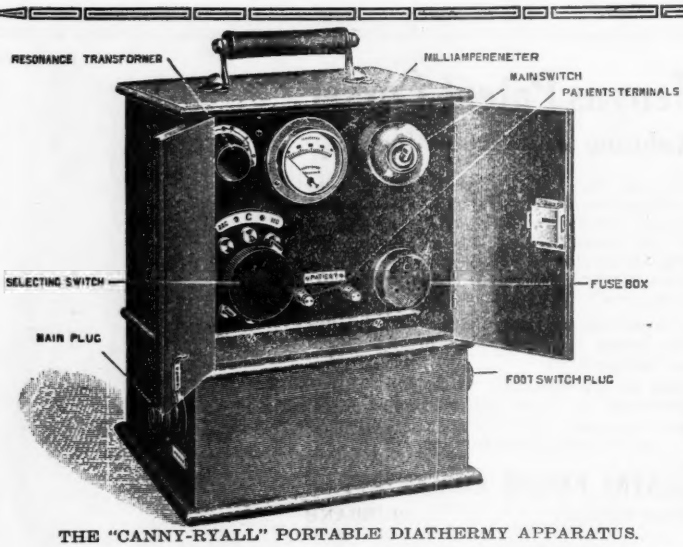

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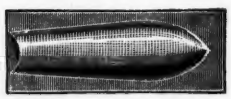

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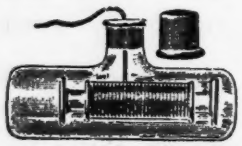

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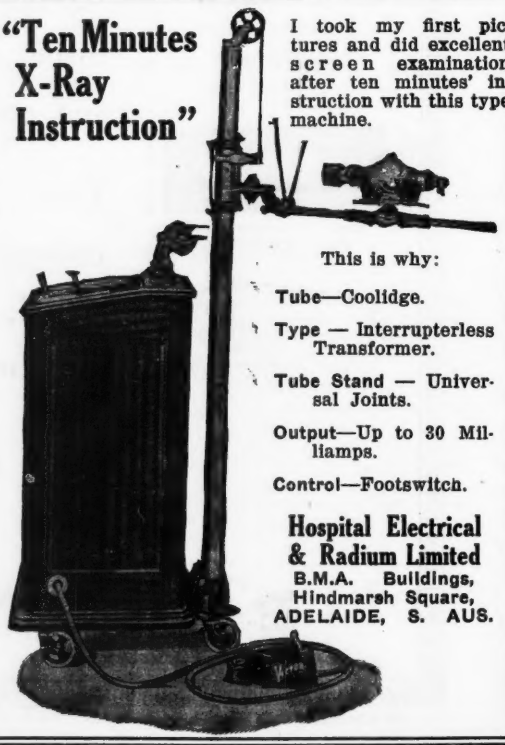

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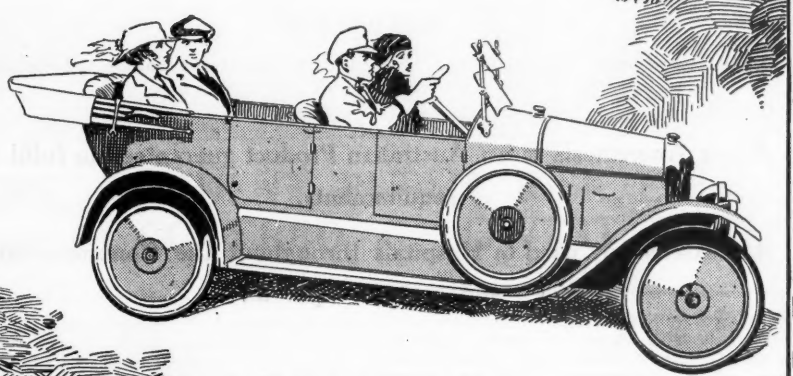

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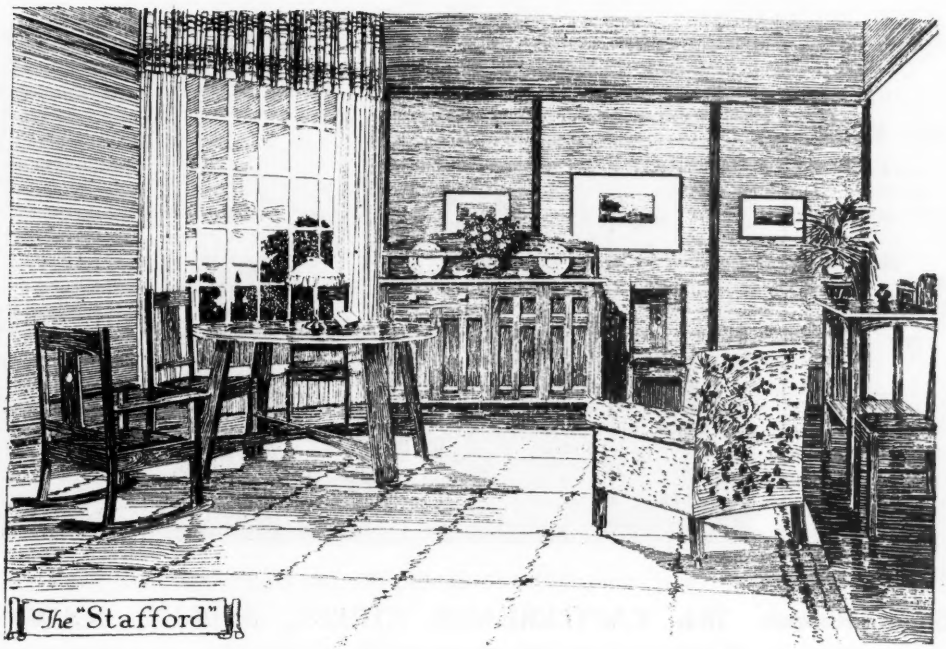

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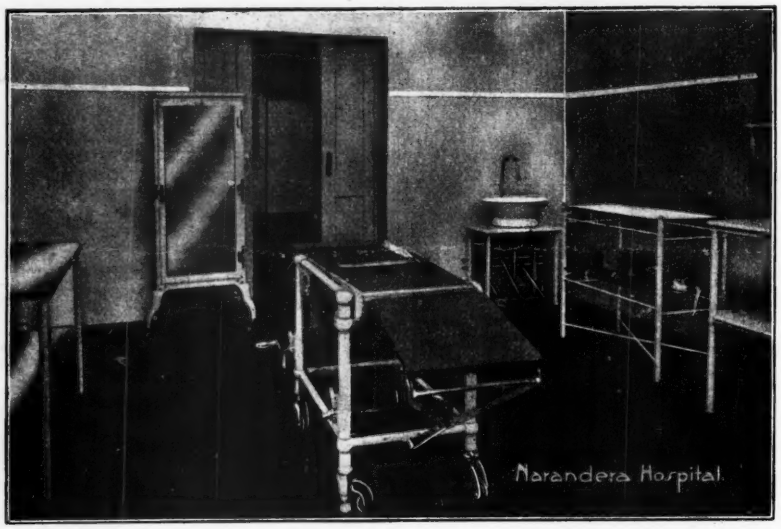

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Department of Health

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Luttinger, Freeman and Saunders have all shown that prophylactic vaccination against whooping cough is highly effective.

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For effective **prophylaxis** three doses should be given at intervals of from five to seven days.

As a first dose, 50 to 500 millions have been suggested by different authors. In respect of **treatment** Luttinger writes:

> "The vaccine treatment at the Whooping Cough Clinic is now the routine. The good results first obtained have continued since the first report (*New York Medical Journal*, May 22, 1915), *i.e.*, we have been able, by the use of the vaccine prepared at the laboratory, to limit the paroxysmal stage to a duration of about three weeks."

He recommends three or four subcutaneous injections—one every other day. The whoop usually disappears between the third and fourth injections. Big doses are to be used.

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Full particulars as to qualifications, etc., may be seen in Commonwealth "Gazette" of December 22 and 29, 1921.

Applications close with the Deputy Public Service Inspector, 527, Collins Street, Melbourne, on January 5, 1922.

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The Medical Journal of Australia.

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APPLICATIONS are invited for the position of ASSISTANT EDITOR OF "THE MEDICAL JOURNAL OF AUSTRALIA" (HALF-TIME). Previous journalistic experience not essential. Preference will be given to Australian graduates under 35 years of age with war service.

Literary ability and a good general knowledge of clinical medicine and surgery, hygiene and pathology and of the conditions of practice essential.

Candidates will be asked to give a reasonable assurance to adopt medical journalism as a career. At present the appointment is half-time, but it is anticipated that full-time employment will be required at a later date.

Information concerning the duties can be obtained from the Editor.

Applications will be received not later than December 31, 1921, by the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, 30-34, Elizabeth Street, Sydney.

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VICTORIA.—(1) Agricultural Town within 50 miles of Melbourne. Takings, £1,150. Sale, £800. Rent, £1 per week. (2) Agricultural District. Takings, £1,826. Appointments, £750. Hospital appointment. Suit surgeon. Sale, £1,500. House (ten rooms), £65 per annum.

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Applications will be received up to March 11, 1922, by the Agent-General for Victoria, Melbourne Place, Strand, London, W.C..

Particulars of duties and other information may be obtained on application to the Secretary, Melbourne Hospital, to whom copies of application and testimonials must be returned by March 1, 1922.

PUBLIC HEALTH TRAVELLING FELLOWSHIPS.

THE INTERNATIONAL HEALTH BOARD is offering FOUR TRAVELLING FELLOWSHIPS IN PUBLIC HEALTH to Australian Medical Graduates.

Each Fellowship is for the period of one year and the successful applicants must undertake to devote that year to the study of some aspect of public health and must undertake on their return to Australia to devote themselves to public health work for five years in the service of the Commonwealth Government.

The course of study must be submitted to the Selection Committee at the time of application and must be referred to and approved by the International Health Board.

The successful applicant will receive:

(1) $1,200 if single and $2,000 if married. The amount is not altered by the fact that the candidate does or does not include travel time in the year of study.

(2) The cost of tuition in the schools determined upon.

(3) The actual and necessary cost of travel from and to Australia, but not expenses of wife or family.

(4) The actual and necessary cost of limited local travel within the United States in connexion with the studies.

(5) The cost of obtaining passports.

Arrangements may be made for portion of the year to be spent in Europe.

Applications are invited from Australian Medical Graduates between the ages of 20 and 35. Each applicant should state his age, marital condition, qualifications, with dates, past medical experience, published research and the course of study abroad which is preferred.

Applications should be addressed to the Director-General of Health, 51, Spring Street, Melbourne, should be accompanied by a photograph of the applicant and must be lodged at the above address on or before January 15, 1922.

W. MASSY GREENE,
Minister of Health for the Commonwealth.

Australasian Trained Nurses' Association

THE following Nurses' Homes are registered by the Council of the A.T.N.A., and receive only Nurses who are members of the Association:

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(3) SISTER TOSHACK'S HOME, 12, Woolcott Street, Darlinghurst. Superintendent: MISS KERSHAW - SLACK. Telephone: William 950.

(4) THE NURSES' CLUB, LTD., 18-20, College Street. Manageress and Matron: MISS LEWIS. Secretary: MISS HILDA E. COX. Telephone: City 3917.

(5) THE MAITLAND NURSES' ASSOCIATION, West Maitland. Matron: MISS KAYE.

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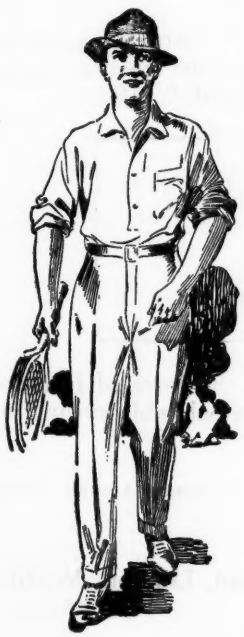

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resulting from bacterial disturbances can be promptly relieved and controlled by the use of

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The use of intestinal disinfectants has been justly discredited, owing to the fact that, hitherto, it has been impossible to administer anything which was at once efficient and non-toxic.

Take the case of Salol, the best available for many years. The dose is five grains (more is unsafe); dissolve this in a quart of water (the average residual fluid of the intestinal tract of an adult) and you get a solution which is too weak to destroy even the most attenuated organisms.

Compare this with Dimol. The dose recommended is two to four pulverettes, say three. Each pulverette contains one grain Dimol, and Dimol is thirty-five times more efficient as a bactericide than Phenol, the active principle of Salol (see test); that is to say, in administering three Dimol Pulverettes, the patient is receiving the bactericidal equivalent of 105 grains of Phenol. Moreover, it is not absorbed, being voided with the fæces, unchanged, after doing its work.

When we are able to repeat this dosage four or five times a day without affecting the appetite or digestion and without action on the mucosa, we can readily understand the excellent results which are obtained by the administration of Dimol in bloody dysentery, typhoid, arthritis, chronic ulcerative colitis, intestinal putrefaction, fermentation, auto-intoxication, etc.

"Examination for phenol in the urine after a week's course of treatment gives a negative result. Tests made for us confirm a co-efficient of 35.0 and the freedom from toxicity claimed by the manufacturers."—"The Lancet," April 9, 1921.

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Sample	Organism	Co-efficient
Dimol	*Bacillus typhosus*	35.0
Phenol	*Bacillus typhosus*	1.0
Salol	*Bacillus typhosus*	0.5

DIMOL has been specially prepared for the Medical Profession in the form of Pulverettes (A coated and B uncoated), in bottes of 50 and 100, and as a palatable syrup for infants and little children in 4-oz. bottles.

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"In treating the cervix and vagina, I use a lamb's wool tampon, with hot Antiphlogistine. This is packed well against the affected parts and held in place by gauze. Portion of gauze is allowed to protrude, as well as the string attached to tampon; and patients given instructions to remove it after allowing it to remain from twelve to fourteen hours."

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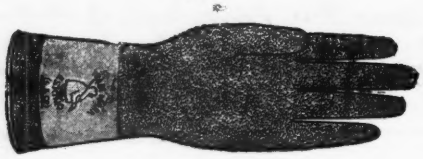

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"THE MEDICAL JOURNAL OF AUSTRALIA"

RECOMMENDS

ARNOTT'S BISCUITS

A UNIQUE AND FITTING TRIBUTE FROM THE OFFICIAL ORGAN OF THE AUSTRALIAN BRANCHES OF THE BRITISH MEDICAL ASSOCIATION.

It is highly pleasing to the Principals of Wm. Arnott & Sons, Ltd., whose Biscuits for half a century have reached the highest standard of popularity throughout Australia, to find that on the occasion of the first Health Week campaign "The Medical Journal of Australia"—the official journal of the British Medical Association in Australia—has published a special article, on Saturday, October 15, endorsing the claims of Arnott's Famous Milk Arrowroot Biscuits to be a perfect hygienically produced food for growing children.

(Extracts from THE MEDICAL JOURNAL OF AUSTRALIA, October 15, 1921.)

When the Analytical Department of THE MEDICAL JOURNAL OF AUSTRALIA was being established, a series of standards for the various classes of infants', invalids' and other foods was drawn up with the collaboration of experts on which the inspection and judgement of these foods would be based. Much careful thought was expended in this preliminary work. It was then determined that the requirements of the JOURNAL in respect to biscuits should be: (*i.*) that scrupulous cleanliness be observed throughout the whole process of manufacture; (*ii.*) that only sound, pure ingredients be used; (*iii.*) that handling be reduced to a minimum; (*iv.*) that the flour used in the manufacture conform to the standards provided in the regulations of the *Pure Foods Act* of the State in which the biscuits are manufactured and sold; (*v.*) that no substance be added that might be deleterious to health; (*vi.*) that the nutritive value of the various forms of biscuits be considerable.

Arnott's have always claimed that Arnott's Famous Milk Arrowroot Biscuits are ideal food "for growing children" and are glad to get this authoritative medical confirmation.

It will be understood that biscuits, being substances containing a large proportion of starch, are unsuitable in all cases for infants under six months of age and that they should be given with a sparing hand, if at all, during the second half of the first and the whole of the second year of life. **Essentially biscuits may be regarded as a suitable food for children and adults.** No expression of opinion can be given concerning the digestibility of any given kind of biscuit, since the only known test is direct experiment and the power of digesting a mixture of starches, proteins and fats varies with almost each individual.

The Factory Buildings.

The inspector sent by THE MEDICAL JOURNAL OF AUSTRALIA was an authority on medical hygiene and biology.

A visit to the factory of William Arnott, Limited, at Homebush, in the neighbourhood of Sydney, has recently been undertaken for us by a special inspector. The factory consists of a group of brick buildings, some of which have been erected at a later period than others. Each building is placed at a sufficient distance from the next one to insure adequate lighting. There is free access of direct sunlight to the intervening spaces, which have been levelled and well drained. The spaces are clean and dry. The buildings are of large area and are well lit. They are provided with cement floors. **Our inspector reports that all parts of the buildings were found by him to exhibit an attractive standard of cleanliness.**

Hygiene of the Factory.

The material from which the biscuits are made are mixed in machines similar to those used in the preparation of dough. The mixture stands in troughs to ripen before further treatment. The machines had been in use shortly before the inspection and all the mixtures had been prepared previously. The machines had been carefully scraped and were quite clean. There were no scraps of dough about the corners or angles. The machines were free from dirt due to oil and flour. The doughs were lying in the troughs, which were all free from signs of old dough. There was no flour on the floor around the machines and the walls were clean and not bespattered with dough.

It has always been the proud boast of Arnott's that biscuits cannot be manufactured under cleaner and more hygienic conditions. Our experts have reviewed the world's leading bake-houses to insure the highest standard of hygienic practice.

The dough is rolled into sheets by means of special machines and these sheets are divided by metal stamps into pieces of the required size and shape. The shaped portions of dough are separated from the trimmings by various ingenious devices. The cut pieces then pass on trays through the ovens. The time occupied in this passage is determined by the temperature of the oven. Many designs of oven are employed at the factory, but in all cases the heating is from below. **The whole of the machinery used in these operations was found to be scrupulously clean. The high standard of hygiene noticeable in all parts of the factory is evidence of the good organization of the concern and the proper training of the numerous employees.**

A comparison between the new arrangement with the use of machines and the older method by manual labour shows that much handling was avoided in the former.

Where machinery will obviate handling, Arnott's will always put it in regardless of cost.

The biscuits are packed in tins or in paper parcels. This is largely done by young women. While some effort has been made to lessen contact with hands, the packing of biscuits still remains a process carried out manually. Rules in respect to washing of hands and to general cleanliness are relied on to provide against any contamination of the biscuits.

We can recommend these biscuits as articles of diet of high nutrient value for children (not infants) and adults. They are carefully prepared and the standard of cleanliness in the factory is of a high order.

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The dose of MOLEVAC is one teaspoonful after or between meals or at bedtime. An immediate effect is not to be expected, and if after three or four days' treatment the desired result is not attained, the dose should be gradually increased till the requirements of the case are met.

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